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# CONTENTS


| | | |
|---|---|---|
| Experimental and Histological Investigation of Gingival Regeneration in Vestibular Surgery | 259 | Gerald P. Ivancie |
| The Application of the Ultrasonic Dental Unit to Scaling Procedures | 264 | Walter N. Johnson<br>John R. Wilson |
| Is Prophylactic Occlusal Adjustment a Justifiable Procedure? | 272 | Robert L. Reeves |
| A Clinical Study of the Effectiveness of the Roll and Charters' Methods of Brushing Teeth | 277 | Glenn H. Curtis<br>Clarence M. McCall, Jr.<br>Hogne I. Overaa |
| Effect on Gingiva and Occurrence of Pigmentation on Teeth Following the Topical Application of Stannous Fluoride or Stannous Chlorofluoride | 281 | Joseph C. Muhler |
| Some Epidemiological Characteristics of Periodontal Disease in a Series of Urban Populations | 286 | A. L. Russell |
| Periodontal Disease in Rural Children of 25 Indiana Counties | 294 | E. M. Benjamin<br>A. L. Russell<br>Roy D. Smiley |
| Periodontal Considerations Associated with Reversible Acquired Prognathism | 298 | Sidney S. Friedman, Jr. |
| A Consideration of Vaccine Therapy in Periodontal Disease | 300 | Basil G. Bibby |
| Comparison of Bacterial Accumulations on Rough and Smooth Enamel Surfaces | 304 | Marjorie L. Swartz<br>Ralph W. Phillips |
| The Distribution of *Pseudomonas Aeruginosa* in Human Saliva and Its Relationship to Gingival Health | 308 | Irving L. Shklair<br>Richard W. Renn |
| Prednisolone in the Treatment of Incapacitating Desquamative Stomatitis | 310 | Edward V. Zegarelli<br>Austin H. Kutscher<br>Herbert F. Silvers |
| Treatment of Infectious Gingivostomatitis and Membranous Stomatitis with Terramycin Mouthwash | 314 | Walter C. Stout |
| Wound Healing | 316 | Edward Lee Howes |
| Editorial | 318 | |
| Announcements | 263, 271, 276, 293, 307, 313, 319- | |

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*and*
**Dr. Grace Rogers Spalding**
*for their pioneer efforts and*
*selfless devotion in behalf*
*of periodontia and the*
*American Academy*
*of Periodontology*

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*THE JOURNAL OF PERIODONTOLOGY* is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

D. P. NACHAZEL, D.D.S.

Chairman, 1957 Scientific Sessions
of the
American Academy of Periodontology

The voluminous details associated with planning the scientific sessions for the American Academy of Periodontology have been capably handled this year by Dr. D. P. Nachazel, of Milwaukee, Wisconsin. Dr. Nachazel is Associate Professor of Oral Medicine at Marquette University School of Dentistry, where he has been teaching for many years. He is past president of the Greater Milwaukee County Dental Society and of the Milwaukee Dental Forum. He conducts a general practice but emphasizes periodontology and full mouth reconstruction.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-SEVEN

# Experimental and Histological Investigation of Gingival Regeneration in Vestibular Surgery

*by* GERALD P. IVANCIE, D.D.S., M.S.,* *Iowa City, Iowa***

THE objective of this investigation was to determine the histological characteristics of alveolar mucosa as compared to gingiva resulting from a surgical attempt to attain gingival regeneration. A review of the literature reveal a number of papers concerned with muco-gingival problems in periodontal therapy such as frenulum position, vestibule depth, repositioning and extension of attached gingiva.[1,2,3,4,5] As of recent date these problems and their therapy have been evaluated on the basis of clinical evidence without histological correlation. It is hoped that this investigation will tend to correlate clinical therapy with basic histologic considerations and evidence.

## INDICATIONS AND PROCEDURE

Several circumstances in periodontal therapy require muco-gingival surgery. These are: (1) pocket eradication in which the base of the pocket is apically or beyond the muco-gingival junction,[3] (2) loss of attached gingiva due to recession and (3) high frenulum attachment. Gingival extension operations were performed for twenty patients in conjunction with eradication of periodontal pockets the base of which extended below or apically to the muco-gingival junction. The following diagrams illustrate the "normal" relationship of the periodontium and a periodontal pocket—the base of which is located apically to the muco-gingival junction (Figs. 1, 2). A clinical view of one of the patients prior to pocket eradication and gingival extension surgery is seen in Figure 3. A clinical view of another patient requiring muco-gingival surgery involving gingival extension and pocket eradication appeared clinically as Figure 4. On such patients after performing a pocket eradication (gingivectomy), a vertical incision was made at each lateral limit of the area to be operated for gingival extension. This incision was carried apically into alveolar mucosa as far as gingival extension was desired. A muco-periosteal flap was then made by blunt dissection exposing periosteum and bone. This flap was then carried approximately one and one-half centimeters apically from its former attachment and sutured to the periosteum (Fig. 5). The wound area was covered with a stiff (zinc-oxide-eugenol) surgical dressing (Fig. 6). The surgical dressing was removed after one week. The post-

*Head, Department of Periodontology, State University of Iowa, Iowa City, Iowa.

**This investigation was supported in part by postdoctoral research fellowships (DF-6077—DF-6806) from the National Institute of Dental Research, U. S. Public Health Service. Read before the Annual Meeting of the International Association for Dental Research, Atlantic City, N. J., March 22, 1957.

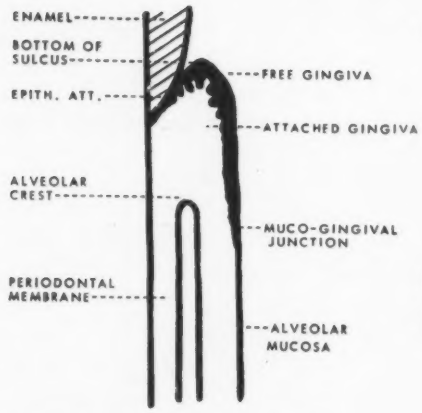


FIG 1. Diagramatic relationship of normal periodontium(After C. Nabers(3))

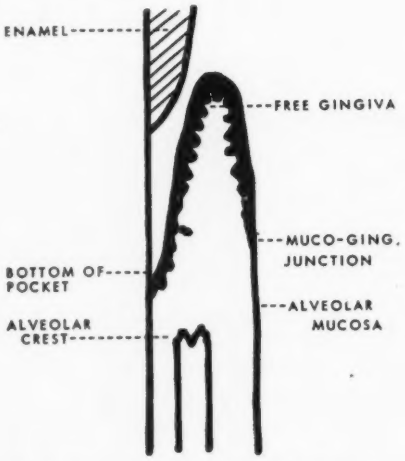


Fig. 2 Diagram illustrating the base of a periodontal pocket- apically to the muco-gingival junction. Pocket eradication would result in the gingival incision located in alveolar mucosa. (After C. Nabers(3))

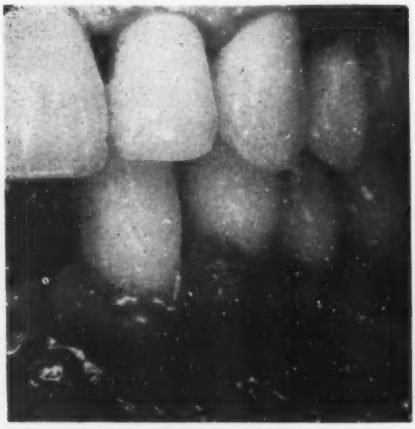

Fig. 3. A patient with a combination of periodontal problems requiring muco-gingival surgery. Periodontal pockets extend beyond the muco-gingival junction. Note the loss of gingiva and high frenum attachment.

Fig. 5. Clinical view of patient after pocket eradication and gingival extension operation by means of a flap operation. Flap was carried apically and sutured (Fig. 4).

Fig. 4. Clinical view of patient with periodontal pockets extending beyond the muco-gingival junction. Note the diminished zone of gingiva.

Fig. 6. Clinical view of wound area following flap operation for gingival extension. Wound area was covered by a stiff (zinc-oxide) surgical dressing for one week.

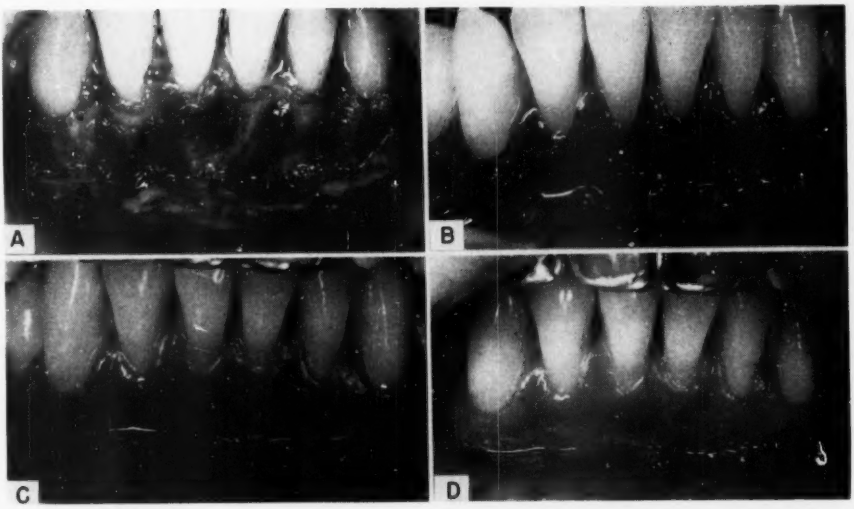


Fig. 7. A. Clinical view postoperatively of wound area after one week following flap operation. Note granulation tissue. B. Clinical view of same area after two weeks. Note degree of healing, level of muco-gingival junction, and degree of gingival regeneration. C. Clinical view postoperatively of former wound area three (3) weeks after gingival extension operation. Note gingival architecture and degree of regeneration. D. Clinical view postoperatively of former wound area (Fig. 5) four (4) weeks after gingival extension operation. Note gingival color, form, texture, and degree of regeneration.

operative area at this time appeared to be healing as characterized by granulation tissue (Fig. 7A). After two, three, and four weeks gingival regeneration appeared clinically evident (Figs. 7B, 7C, 7D). Biopsies were taken from the operative sites above the muco-gingival junction at intervals of one (1), six (6) and eight (8) months postoperatively. For purposes of evaluating alveolar mucosa and gingiva of each patient a control preoperative biopsy was taken from a clinically "normal" area as close to the operative site as conditions permitted. The biopsied area included alveolar mucosa and gingiva (Figs. 8, 9). Biopsies were fixed in 10% formalin. In

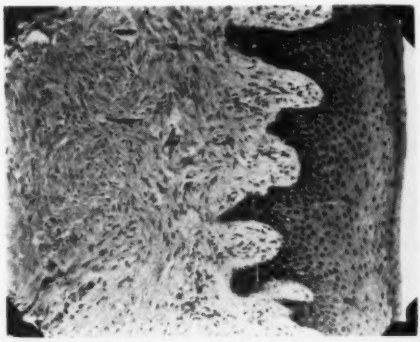

Fig. 10. Biopsy from former wound area indicating gingival regeneration in progress after one month. Note incomplete keratinization, low epithelial ridge formation resembling mucosa, and immature type of connective tissue characterized by lack of orientation of collagen fiber bundles.

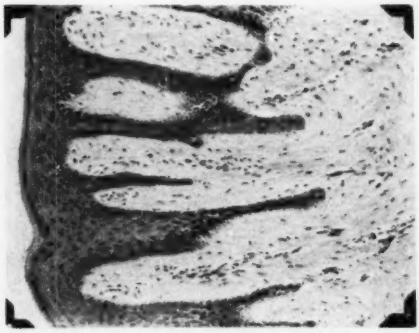

Fig. 11. Biopsy from former wound area indicating complete gingival regeneration after eight months. The characteristics of this new tissue are entirely that of gingiva.

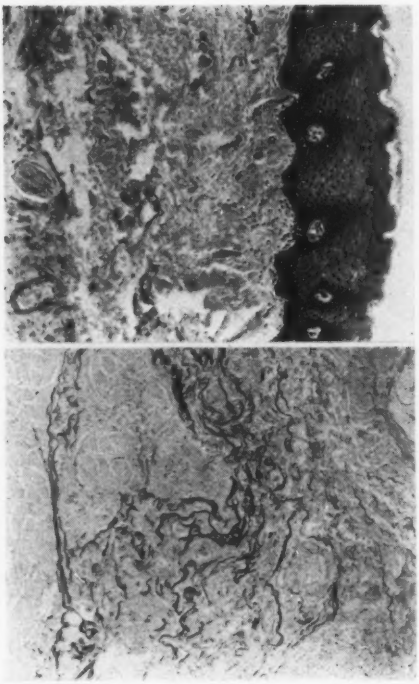

Fig. 8. A—(upper) Biopsy specimen (control) of "normal" alveolar mucosa adjacent to the area operated. Note incomplete keratinization, low epithelial ridge formation, and orientation of connective tissue. B—(lower) Biopsy specimen (control) "normal" alveolar mucosa adjacent to the area operated. Note numerous elastic fibers (orcinol).

addition to hematoxylin and eosin an orcinol elastic tissue stain was employed to determine the presence or absence of elastic tissue fibers in the area of gingival regeneration.

## RESULTS

After one (1) month postoperatively (Fig. 10) the tissue resembles mucosa with respect to epithelium rather than gingiva. Although some epithelial ridge formation is in evidence as well as beginning partial keratinization it has not assumed the characteristics of the control for "normal" gingiva.[6] The connective tissue is of rather immature type without good orientation of the collageneous fiber bundles. The connective tissue is more cellular than normal

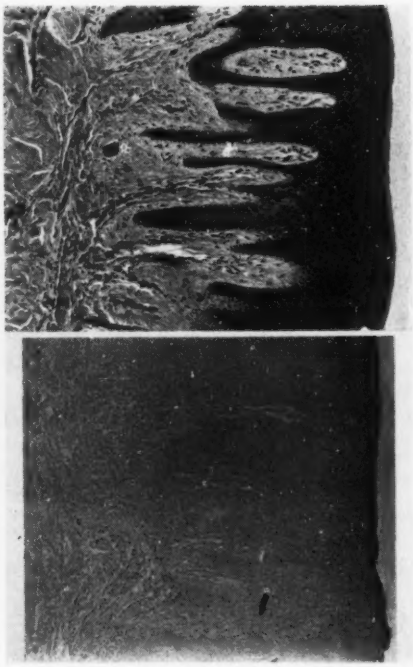

Fig. 9. A—(upper) Biopsy from clinically "normal" gingiva adjacent to area operated. Note well keratinized surface of epithelium, regular ridge formation, and dense connective tissue. B—(lower) Biopsy from clinically "normal" gingiva adjacent to the area operated. Note absence of elastic fibers (orcinol).

gingiva. Six (6) months after surgery the biopsies reveal complete regeneration of the characteristics of normal gingiva. At the end of eight (8) months after surgery

Fig. 12. Biopsy from former wound area indicating complete gingival regeneration. Note absence of elastic fibers.

the characteristics of the tissue is entirely that of gingiva. The epithelium shows long and rather regular ridges with the surface keratinized (Fig. 11). The subepithelial connective tissue shows good orientation of the collageneous fiber bundles.[7] The elastic fibers which are prominent in the alveolar mucosa (Fig. 8B) were not found in the regenerating gingival tissue (Fig. 12). In several specimens small amounts of elastic fibers were evident. Their presence is believed due to incomplete dissection during surgery.

## DISCUSSION

The importance of this investigation has to be viewed from the functional point of view. In response to a surgically created new environment newly regenerated gingival tissue is formed. Gingival regeneration occurred in an area that formerly existed as alveolar mucosa. Epithelial ridge formation, keratinization, and orientation of mature collagen may be viewed as functional adaptation.[8] This study shows the possibility of complete gingival regeneration.

## SUMMARY

Histological evidence from this study reveals that regeneration of gingival tissue had occurred within eight (8) months postoperatively after surgery. The clinical application of this investigation should be obvious.

The author wishes to thank the Colorado Dental Foundation for taking and processing the photomicrographs used in this article.

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## SURVEY TO BE MADE BY THE AMERICAN COLLEGE OF DENTISTS

The National Opinion Research Center of the University of Chicago, at the request of the American College of Dentists, and in cooperation with the Walter G. Zoller Memorial Dental Clinic, is currently engaged in a study of procedures and problems of dental practice. Personal interviews with a nationwide sample of active, practicing dentists are now being conducted. Results of the study, which will be useful to the entire dental profession will be made generally available in summary terms toward the end of the year. The main interest of the study is in the procedure and problems of dental practice, including their preventive aspects.

# The Application of the Ultrasonic Dental Unit to Scaling Procedures*

*by* WALTER N. JOHNSON, D.M.D., M.S.,** AND JOHN R. WILSON, D.D.S.,***
*Columbus, Ohio*

SINCE Catuna first called attention to possible application of the ultrasonic cutting principle in dentistry,[1] the profession has exerted maximum effort to evaluate the procedure. The dental literature contains several reports pertaining to the technical aspects of the ultrasonic dental unit.[2, 3, 4, 5] Editorial comment expressed in professional dental journals has pleaded for thorough testing before the ultrasonic principle is used in routine practice.[6, 7, 8, 9, 10, 11, 12, 13] The experimental results pertaining to instrumentation with an ultrasonic dental unit in operative dentistry have been encouraging.[3, 4, 14, 15, 16, 17, 18, 19, 20, 21, 22] Biological evaluation of the new instrument has also been encouraging with the majority of the investigators reporting no more severe pulpal changes in teeth ultrasonically treated than in teeth treated with conventional rotary instruments.[16, 23, 24, 25, 26] One group of investigators has reported severe adverse biological results in the pulpal tissues of guinea pigs after ultrasonic instrumentation.[27] Of all this early work, only one report deals with the application of the ultrasonic principle to calculus removal. This work was reported by Zinner in the Proceedings of the Fourth Annual Conference on Ultrasonic Therapy in 1955.[19] In this paper Zinner reported planning calculus deposits from teeth with the ultrasonic tip in the presence of water. This was done both in-vitro and in-vivo. Zinner reported that there was very little bleeding and no adverse subjective clinical symptoms. The report was very encouraging.

The purpose of the study reported here was to demonstrate the effectiveness of ultrasonic energy when applied to the removal of calculus and other deposits from the teeth. This demonstration included a comparison of the ultrasonic method to routine hand scaling methods. A second part of the problem was to develop and demonstrate effective scaling instruments with which to scale all surfaces of the teeth. The Cavitron Ultrasonic Dental Unit was used in these experimental procedures.

## EXPERIMENTAL PROCEDURES

An extracted tooth with heavy deposits of calculus as cleaned with a scaling tip similar to the ultrasonic scaling tip number 2b (Fig. 12). For all scaling procedures during the investigation the "output" on the Cavitron Unit was set at "70" and it was tuned for maximum cavitation. The calculus was easily removed amidst a spray of water. Stain was also effectively removed. No perceptible vibration could be felt either in the transducer handpiece or in the extracted tooth. In the absence of water some calculus could be removed but both the tooth and the tip became extremely hot from friction.

A simulated mandibular arch of extracted teeth was set up in a dentoform. These teeth exhibited large amounts of calculus, stain, and remnants of periodontal membrane. Each surface of every tooth was scaled using a variety of instruments. The calculus and stain were easily removed but when an attempt was made to remove the remnants of the periodontal membrane extreme difficulty was encountered. The softer tissue seemed to be displaced before the vibrations of the ultrasonic instrument.

*This paper is based on a thesis submitted in partial fulfillment of the requirement for the Master of Science Degree at the Ohio State University.

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***Professor of Periodontology, The Ohio State University.

The opinions or assertions contained in this article are the private ones of the writers and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

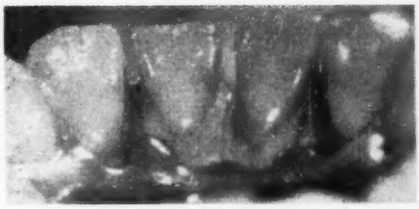

Fig. 1. Lingual view of the lower anteriors of a 37 year old female before scaling.

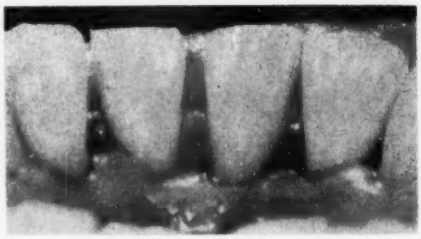

Fig. 2. Teeth shown in figure 1 after scaling with ultrasonic energy.

This part of the experiment demonstrated the versatility of the instrument as applied to scaling all surfaces of the teeth.

Ten clinical patients with heavy calculus were scaled using ultrasonic technique. Figure 1 shows the lingual of the lower anteriors of a 37 year old female before scaling the ultrasonic energy. Figure 3A shows a radiograph of these teeth before scaling. This case was characteristic of the clinical cases treated. The post scaling view of these teeth is shown in Figure 2. Clinical inspection of the scaled teeth conducted with the aid of adequate light, mouth mirror, explorer, air blast, and radiographs (Fig. 3B) revealed no residual deposits. These six teeth were scaled in eighteen minutes. During the instrumentation there was slight hemorrhage from the gingiva. The patient was not anesthetized and reported no disagreeable sensations during any of the procedures. Water was delivered to the handpiece from the heating unit of a standard dental unit rather than from the ultrasonic dental unit.

Since it had been demonstrated that ultrasonic energy was effective in eliminating deposits, the next step was to compare the effectiveness of ultrasonic scaling with that of conventional hand scaling procedures in deep periodontal pockets. A 42 year old male with six condemned lower anteriors (Fig. 4) was selected for this phase of the experiment. A gingival flap was laid to demonstrate the existence of subgingival deposits (Fig. 5). The flap was repositioned, sutured, and allowed to heal for one week (Fig. 6). Figure 7 shows the lingual view. Pre-scaling radiographs are shown in Figure 8. The subgingival root surfaces of the mandibular left central, lateral, and cuspid teeth were scaled with conventional hand scalers while the right central, lateral, and cuspid were scaled with the ultrasonic unit. Local anesthesia was administered prior to scaling. Constant

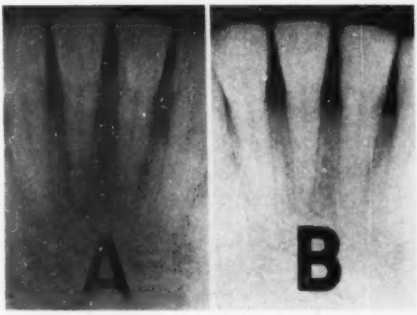


Fig. 3. Radiographs of the teeth shown in figure 1. "A" before scaling and "B" after scaling with ultrasonic energy.

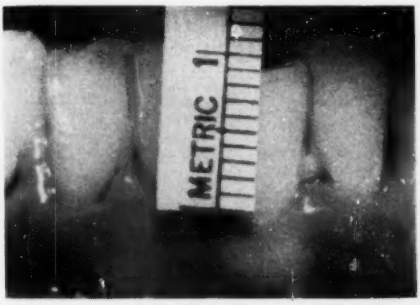


Fig. 4. Lower anteriors of 42 year old male before surgery and scaling. Metric rule indicates the level of the gingival crest.

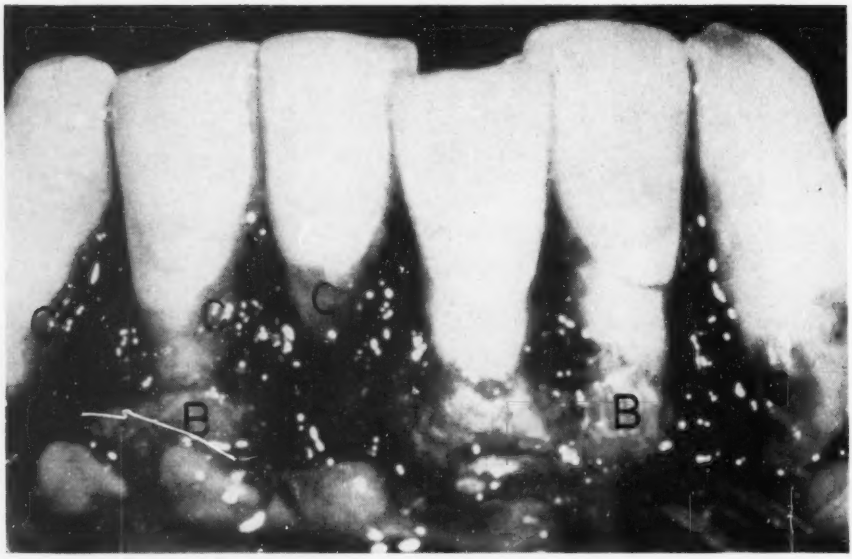


Fig. 5. Teeth shown in figure 4 with labial flap laid. "C" calculus and "B" bone.

aspiration was required during the ultrasonic scaling to control the excess water. No aspiration was used during the hand scaling. Forty minutes was allowed for each procedure in which to remove all the subgingival deposits. If the subgingival deposits was completely removed prior to the forty minutes, then the remaining time was to be spent scaling the supragingival surfaces. After scaling, the teeth were extracted, examined and photographed.

In-situ photographs of the scaled teeth are shown in Figures 9 and 10. Photographs of the extracted teeth are shown in Figure 11. All of the subgingival deposits were removed from the teeth except in two instances. A piece of calculus remained on the mesial root surface of the mandibular right central which was ultrasonically scaled and another piece remained on the distal root surface of the mandibular left central which was hand scaled. More deposits were removed during the forty minute period with the ultrasonic instruments than with conventional scalers. In addition, the ultrasonic method was effective in the removal of stain from the lingual coronal surfaces. Some gouging or scratching on the root surfaces was seen on each tooth scaled. This marking was more pronounced on the teeth scaled with the ultrasonic method. The most severe damage was a cut into the dentin made by the ultrasonic instrument on the root of the mandibular right central incisor. The patient appeared to be more relaxed during the ultrasonic scaling and expressed a preference for that method. He cited the absence of tugging and scraping associated

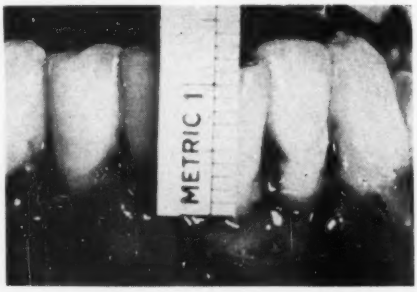


Fig. 6. Teeth shown in figure 5 following one week's healing. Metric rule indicates new level of the gingival crest.

Fig. 7. Lingual view of the teeth shown in figure 4 after one week's healing following flap operation.

with hand scaling. There was less hemorrhage associated with the ultrasonic method than with the hand scaling.

Seven scaling tips were designed to adequately clean every tooth in the mouth. The application of these instruments is presented for suggested use only since it is understood that each dentist may modify both the instruments and method of use to his own requirements.

Ultrasonic scaling tip number 1 (Figs. 12, 13) was designed to reach the difficult proximal areas of a tooth near the contact point. This scaler has a thin tapering blade which facilitates cleaning these areas.

The ultrasonic scaling tip number 2a (Fig. 12) was designed to scale the labial aspects of the roots of the mandibular and maxillary anteriors and bicuspids. The buccal surfaces of bicuspids may be scaled with tips 3a or 3b if a small mouth limits access. The number 2a is versatile in that the reverse side of the tip (the convex side) can be used to clean the coronal lingual aspect of the crowns of the mandibular and maxillary anterior teeth.

The ultrasonic scaling tip number 2b (Fig. 12) is the complement to 2a. This tip is designed to scale the lingual aspect of the roots of the mandibular and maxillary anteriors and bicuspids. The reverse side of this tip may be used to clean the coronal labial surfaces of the mandibular and maxillary anteriors. Both 2a and 2b are used with a push motion. The instrument is guided down the root surface towards the apex, thereby pushing the calculus ahead of the point. Enough water is used during scaling so that the pocket is adequately flushed free of calculus. A pulling motion with the scaler will function with some success, but was not found to be as efficient as a push motion. Care must be taken to maintain a light touch during scaling to avoid gouging the root surface.

Ultrasonic scaling tip number 3a (Fig. 12) is used to scale the buccal surfaces of

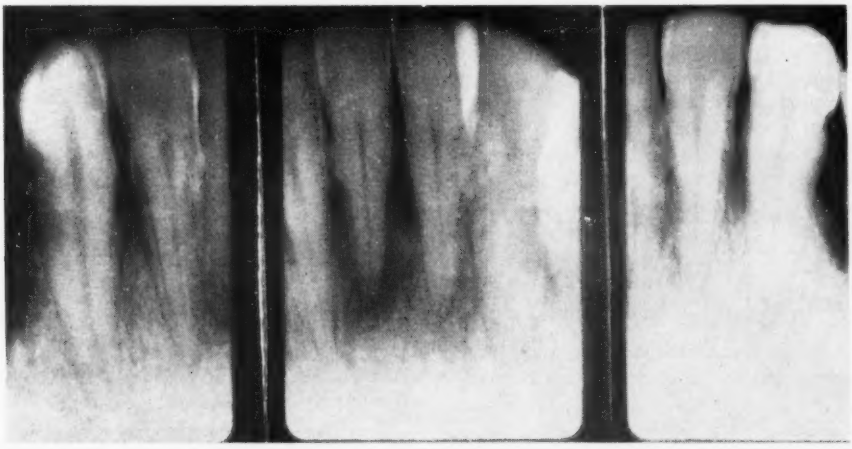

Fig. 8. Radiographs of the teeth shown in figure 4 before scaling.

the mandibular right molars and maxillary left molars. Number 3a is also used to scale the lingual surfaces of the maxillary right molars and mandibular left molars.

Ultrasonic scaling tip number 3b (Fig. 12) is the complement to 3a. Number 3b is designed to scale the lingual surfaces of the mandibular right molars, and maxillary left molars. It is also used to scale the buccal surfaces of the maxillary right molars and the mandibular left molars.

Ultrasonic scaling tip number 4 (Fig. 12) is designed to scale the interproximal root surfaces of all posterior teeth including the distal of the last molars. The scaling tip number 1 is used in conjunction with number 4 to thoroughly clean the proximal surfaces of these posterior teeth.

Ultrasonic scaling tip number 5 (Fig. 12) can be used on interproximal root surfaces of the anterior teeth.

In our hands, the most efficient scalers are ultrasonic scaling tips numbers 1, 2a, 2b, and 4. This efficiency is the result of an elliptical motion that the working end of the tip describes during function. Since tips numbers 3a, 3b and 5 are more rigid the elliptical motion is reduced, hence the reduction in scaling effectiveness.[28]

## DISCUSSION

The experiments reported here confirmed the preliminary reports of Zinner with respects to ease of calculus removal, lack of hemorrhage, and patient acceptance. It was found that the minimum amount of water required for the ultrasonic procedure still resulted in an overflowing field which could be controlled only with the aid of a "vacudent." By using warm water from a dental unit a more desirable temperature was available rather than the cold water delivered by the ultrasonic unit.

The mechanism by which calculus is removed was not determined in this study. There is no direct evidence which links cavitation with the removal of calculus. It was found that it was necessary to touch

Fig. 9. Labial view of the teeth shown in figure 4 after scaling. The right cuspid, lateral, and central were ultrasonically scaled and the rest were hand scaled.

the calculus with the scaling tip to cause its detachment from the tooth. When the tip touched one area of the calculus it was not unusual for a much larger area to come free from the tooth. Merely holding the instrument close to the calculus so the cavitation water played over it was not sufficient to remove the calculus. Calculus could be removed in the absence of water but the heat generated from the friction of the rapidly moving point in contact with the tooth was excessive. The use of water facilitated the removal of calculus and kept the tooth and tip cool during the procedure.

The mechanism by which stain is removed by the ultrasonic method is also unanswered. Stain can be effectively removed from a crevice that has smaller dimensions than the scaling instruments. This may be due to energy released by cavitation.

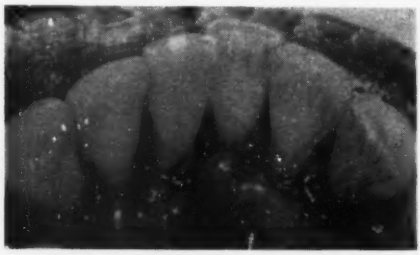

Fig. 10. Lingual view of the teeth shown in figure 9 after scaling. Note the absence of stain on the coronal surfaces of the right cuspid, lateral, and central which were ultrasonically scaled.

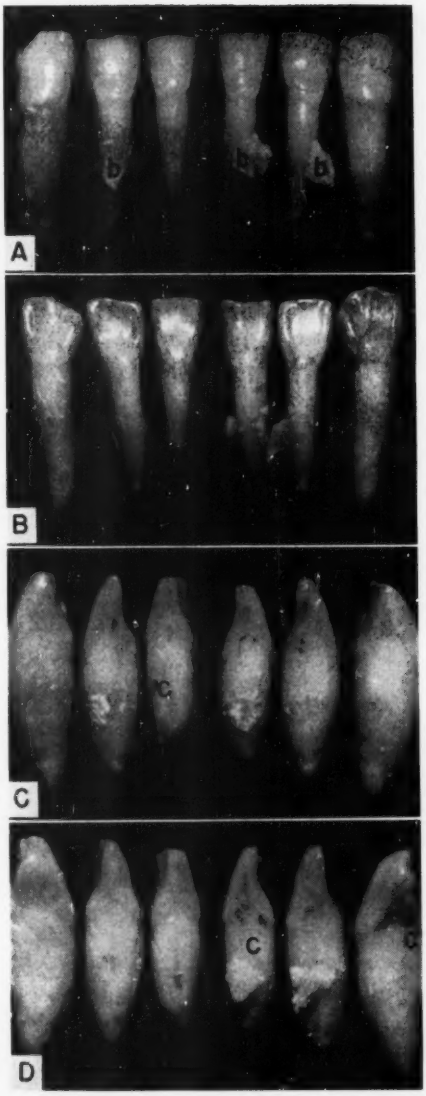


Fig. 11. The teeth shown in figure 4 following scaling and extraction. The three teeth on the left were ultrasonically scaled and the rest were hand scaled. "c" calculus and "b" bone.

Only the lightest pressure was necessary to remove deposits. Heavy pressure actually slowed the removal of the calculus.

After calculus removal the cemental surface as viewed under a dissecting microscope appeared as though it were newly formed with gentle undulations and small pores. The surface was smooth but not flat.

Since soft tissue is apparently immune to injury by the scaling tip, scaling to the depth of the periodontal pocket may be accomplished without damage to the periodontal attachment. Other soft tissues of the oral cavity would be similarly immune to accidental exposure to the scaling tip while the instrument is in function.

The comparison of the ultrasonic to hand scaling showed that neither method was superior in the quality of the scaling since both instruments demonstrated good results subgingivally. Since more deposits were removed in the allotted time with ultrasonic scaling than with conventional scaling, the ultrasonic scaling method was judged superior. This, coupled with the removal of stain weighed heavily in favor of the ultrasonic method. Some time was lost in the hand scaling procedures because the patient was permitted repeated rinsings.

The experiment demonstrated the fact that the cementum and even dentin of the root can be injured with the ultrasonic technique. It is believed that if the scaling tips are kept flat or gently rounded on the working end and if only light pressure is employed, gouging will be reduced to a minimum. During function the scaling tips become worn and actually sharpened. A quick dressing with a carborundum stone is all that is required to restore the original flat end to the tip.

## SUMMARY AND CONCLUSIONS

Experiments covering both in-vitro and in-vivo applications of the ultrasonic dental unit to scaling procedures were presented. The ultrasonic method was compared to hand scaling for efficiency of calculus removal versus time required.

Seven ultrasonic scaling tips were designed and a discussion of their application to scaling procedures was presented. The procedures were illustrated with photo-

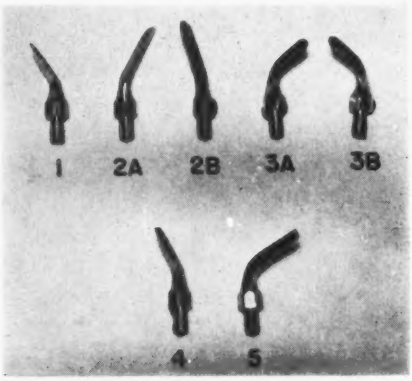


Fig. 12. Ultrasonic scaling points. Numbered as indicated.

graphs showing the relative position of the instruments to a typodont.

The conclusions reached from the experimentation are as follows:

1. The ultrasonic scaling technique will remove calculus.

2. Only light pressure is necessary to scale with the ultrasonic technique.

3. Stains are effectively removed with the ultrasonic scaling technique.

4. Cementum under calculus is virtually unharmed after scaling if the working end of the instrument is flat instead of sharp and only light pressure used during scaling.

5. Comparatively little hemorrhage is associated with the ultrasonic scaling procedure.

6. The adequate removal of calculus is accomplished more rapidly with the ultrasonic scaling method than with conventional scaling methods.

7. Calculus removal is possible using the ultrasonic method without water but is impractical due to heat generated from friction.

8. When water hits the vibrating scaling tip a mist is generated which makes vision difficult.

9. A preference was registered by patients for ultrasonic scaling when compared with hand scaling.

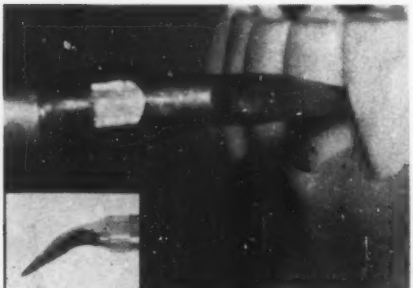

Fig. 13. Ultrasonic scaling instrument number 1.

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# Is Prophylactic Occlusal Adjustment a Justifiable Procedure?

*by* ROBERT L. REEVES, D.D.S.,* *Los Angeles, California*

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**Quotation authenticated by Dr. Paul N. Baer.

THE increasing prolificness of the writers on occlusion is apparent to all students of this subject. Over two hundred papers a year dealing with some aspect of occlusion are currently being published in the English language. Not all individuals realize, however, the venerability of the literature on occlusion, as the following quotation will show:[10]

*Teeth and Gums*
*Thirty white horses upon a red hill,*
*Now they tramp, now they clamp,*
*now they stand still.***

This discussion will be limited to the question of occlusal adjustment in the absence of pathology. The concept of prophylactic occlusal adjustment has been accepted by a number of writers during the past few years,[4, 12, 19, 20, 22, 23, 26, 32, 33] while others have considered such an approach unjustifiable.[7, 9, 15, 21, 24, 25, 28, 29, 30]

To clarify the question and to further limit the scope of this discussion certain exceptions should be noted. Occlusal adjustment by orthodontic means, followed perhaps by occlusal grinding, will not be considered, nor will situations in which a number of occlusal surfaces must be restored through operative procedures or by means of fixed or removable prosthesis.

The relationship of occlusion to pathology should be realized, whether a preventive or a therapeutic approach is to be taken. It is certainly incumbent upon those who attempt to prevent disease to know what disease they are attempting to prevent.

Occlusal disturbances may affect either the temporomandibular articulation, or certain of the periodontal tissues, or both. That pathology of the joint and musculature may be caused by occlusal problems is a fact generally recognized. The list of periodontal ills that have been attributed to "traumatic occlusion" is so long and all inclusive that, if the list were correct, it would be difficult to examine any mouth without seeing some disturbance on which to blame the occlusion.

The role of occlusion in periodontal disease has been the subject of much attention. Discussion of past and present thought, along with adequate references, is presented in the recent texts on periodontology.[1, 3, 6] Continuing experimental evidence[2, 5, 31] has been added to our knowledge, even since the recent publication of these books.

No attempt will be made here to comprehensively review the opinion and evidence now available, but a resumé will be given.

1. Changes may occur in tissues of the supporting apparatus of a tooth from forces applied to the tooth. The tissues that may be affected are the cementum, the periodontal membrane and the alveolar bone. The changes may either be pathological in nature or compensatory. The amount or magnitude of force is not the only factor influencing these tissue changes; the direction of force, duration of force and frequency of force also play a part.

2. Occlusal overloading is not an etiological factor in gingivitis or gingival hyperplasia. Migration of teeth, sometimes a result of occlusal forces, may bring about impaction and impingement of food against the marginal gingiva and interdental papilla, with subsequent gingivitis. The etiologic factor here is food impaction and impingement, not trauma from occlusion.

3. Occlusal overloading will not initiate pocket formation. Local irritants such as calculus, food impaction and bacterial activity are necessary to induce apical migration of the epithelial attachment. Rapidity and depth of pocket formation may be accelerated by concomitant traumatic damage from occlusion, however. The significance of this differentiation is that pockets are neither prevented nor treated by occlusal adjustment.

4. Occlusal overloading will not cause gingival recession, gingival clefts, or resorption of the alveolar crest. As stated previously, trauma from occlusion is apparently not a primary etiological factor in apical proliferation of the epithelial attachment, which would be necessary before gingival recession or clefts could occur. In spite of experimental evidence so far to the contrary, an opinion, based upon clinical experience, is still held by some individuals that periodontal atrophy or gingival recession may be caused directly by trauma from occlusion.

Resorption of the crest of the alveolar bone on the labial, buccal, or lingual of a tooth could conceivably be caused by occlusal force if the bony plate were extremely thin. In such an instance, resorption which would ordinarily only enlarge the alveolus might destroy the bone through its entire thickness. It should be emphasized, however, that the usual pattern of bone loss from occlusal traumatism is one of resorption of the "alveolar bone proper," as evidenced on the roentgenogram by a widening of the periodontal membrane space.

The relationship of occlusion to pathology has been considered from two viewpoints. The first of these is primarily concerned with the relationship of cusps, joint and musculature and other parts of the "stomatognathic system." Disharmony between any of these parts is viewed with alarm, although the exact consequences of such a disharmony are not always made clear.

McCollum[18] has considered what he calls "malarticulation" as pathology in itself, writing,

"But there is a subtle pathology of function in nearly every mouth, due entirely to the malrelations between the opposing teeth themselves and between the interdigitation of the teeth and jaw motions."

More recently Granger[11] has said,

"When a disharmony exists between the teeth and joint, there is a constant struggle going on between the two. Usually the joint wins the battle and the periodontium is destroyed with subsequent loss of the teeth. Sometimes the teeth win the battle and the joint is injured. Occasionally they both succeed in injuring each other."

Further illustrative of this point of view is the writing of Lauritzen:[17]

"Occlusal disharmonies and other functional disturbances of the bite should be recognized and diagnosed routinely in order to institute a treatment producing a well functioning bite. The factors constituting the complete masticatory organ, which include the cheeks, lips, tongue, single teeth, the rows of teeth, the mucous membranes, all periodontal structures, jaws, muscles, ligaments and temporomandibular joints, are, during function, stimulated most favorably when they are in a specific harmonious mutual relation, or physiological equilibrium."

Those who adhere to this approach are often inclined to attempt to prophesy disease and to adjust occlusion prophylactically, either by grinding or by "reconstruction."

The second school of thought gives primary attention to the tissues that may be involved if damage should occur. The presence of two important variables support this viewpoint: (1) Force cannot be exerted, and pathology possibly result, except when the teeth are brought into contact. Contact during *mastication* is too light and fleeting[16] and occupies such a small proportion of the day that it is seldom important in a traumatogenic sense. It is therefore the "afunctional" movements (such as bruxism and clenching) that are the most significant. The extent to which individuals indulge themselves in these habits differs extensively; (2) Tissue adaptability varies so greatly that its reaction to occlusal forces cannot be foretold.

Following this course of thought the term "occlusal inbalance" is used to denote an occlusal relationship under which the tissues are breaking down, rather than an uneven articulation or premature contact.[29]

To supplement the opinions available in the literature, inquiry was directed to several periodontists in the United States, all of whom are university teachers in the field. The questions asked were:

*Do you feel that occlusal adjustment is routinely indicated in the absence of demonstrable temporomandibular joint disturbances or periodontal disease affecting the supporting tissues of the teeth?*

*If you believe that occlusal adjustment should not routinely be done in the absence of pathology do you feel that*

*such an approach might be indicated occasionally, and if so what are the indications?*

Of the ten who replied, seven[8, 15, 21, 24, 25, 29, 30] felt that routine occlusal adjustment in the absence of related pathology was not a justified procedure.

Among those holding a contrary view to the majority, one[14] gave a qualified "yes" to the first question:

> It is *my* opinion that premature contacts should be corrected when noted even before any damage results from it. This I consider "prophylactic." I also recommend adjustment of all high restorations and obvious facets worn in natural teeth even though there is no demonstrable temporomandibular joint or periodontal disease. I do not believe it is justified, however, to perform major occlusal adjustments on every adult. It seems to me that more harm than good *can* result by extensive re-contouring of natural teeth. We do not therefore recommend occlusal equilibration on a grand scale for patients unless they do have periodontal disease or temporomandibular joint disturbances which might be associated with malocclusions.

Further opinion on the "yes" side held:[19] ". . . I believe as we become more experienced we can see potentiality as well as actuality. If such is the case, an occlusion that we know is going to cause trouble when other etiologic factors become strong enough should be treated. The amount of treatment, again, must depend on the judgement and skill of the operator."

Opposition to routine prophylactic occlusal adjustment is based upon the following:

1. It is the misuse of the occlusion through tensional habit patterns, modified for better or worse by the adaptability of the tissues themselves, that inflicts the damage, not the position and contact relationships of the teeth per se.

2. Prediction that either temporomandibular joint or periodontal disease will result from occlusal disharmony is impossible. An interesting point in this regard was made by Thomas, who wrote,[30] "In my own mouth I refused such treatment as an undergraduate dental student twenty-two years ago. Today my radiographs show the alveolar crests practically unchanged from the way they looked in 1933. Had the occlusion been 'equilibrated' I would probably be saying that the reason the bone level is so good today is because I had 'prophylactic occlusal adjustment.'"

3. Trying to make all patients' occlusions fit a preconceived template of so-called "ideal" occlusion is neither practical nor rational. The concept of "normal" occlusion has been derived from orthodontics and prosthodontics. This idea represents a step in the evolution of thought in these fields and its development is completely understandable. The degree of rarity of *natural* articulations that even outwardly approach this "normal" is well known to all dentists. Hellman[13] has computed the theoretical maximum number of contacting points and surfaces of thirty-two teeth as 138 positions of contact. Schireson,[27] in an informal study using articulating wax and paper, found that for natural dentitions with most teeth present the usual range of actual contacting positions was only one-third to one-half of the theoretical maximum.

The "norm concept" becomes even more untenable when it is examined against the background of human evolution. Hellman has further pointed out:

> (1) That the occlusion of the teeth in man was attained by extensive transformations in form, complications in structure and elaboration in pattern.
>
> (2) That the process of evolution in the teeth and their occlusion is not at an end but is continuing to operate in the dentition of modern man.
>
> (3) That the trend in man is toward simplification in forms of teeth and reduction in number of occlusal contact units.

It can thus be seen that present-day man has not inherited a fixed, perfected occlusion, any deviations from which must be considered a deformity.

4. Occlusal adjustment has certain potential hazards. One of the greatest of these is the destruction of what Fox has termed the *negative occlusal sense*: "those proprio-

ceptive and exteroceptive qualities to which a patient has become so accustomed that he is unaware of the reflexes and sensations associated with his teeth or jaw movements."[9] Many dentists are aware of the situation in which a patient who had no complaints about his occlusion (and who in fact did not even know he had an occlusion!) becomes acutely aware of minor disparities following occlusal adjustment. This negative occlusal sense is intrinsic in all people and is destroyed to a lesser or greater degree by any change in occlusal relationships.

Exceptions were noted by those who opposed routine occlusal adjustment in the absence of pathology:

1. The relieving of gross prematurities, believed to be causing bruxism or other grinding compulsions;

2. The practice of correcting incisal aberrations for esthetics;

3. As noted previously, occlusal grinding in conjunction with orthodontic procedures or along with operative or prosthetic procedures requiring the restoration of a number of occlusal surfaces.

## SUMMARY

1. The question of the justification of occlusal adjustment in the absence of related pathology is controversial. (The etiological role of occlusion in periodontal disease was reviewed.)

2. One viewpoint on occlusion focuses upon the static and active relationships of the various parts of the stomatognathic system. Pathology is inferred from any "disharmonies" that can be seen.

3. Another viewpoint considers the reactions of the tissues themselves to be of primary importance:

a. Almost all pathology results from tensional habits rather than from mastication and swallowing. Tensional habits vary greatly in individuals, both to type and frequency;

b. Tissue adaptability to occlusal forces is so variable that prediction of disease cannot be made.

4. In dealing with natural dentitions a concept of "normal occlusion" is neither practical nor rational.

a. Occlusions rarely, if ever, approach this concept of "ideal."

b. From the evolutionary standpoint the occlusion of man is ever-changing. It is neither static nor "perfected."

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## INSTITUTE OF PATHOLOGY ANNOUNCES COURSE IN ORAL PATHOLOGY

The Dental and Oral Pathology Branch of the Armed Forces Institute of Pathology, Washington, D. C., will present its annual course in "Pathology of the Oral Regions" during the period March 24-28, 1958.

The course is designed to familiarize both physicians and dentists with the clinical features and histologic aspects of diseases of the oral regions. This year emphasis will be placed on malignant neoplastic disease. Color television will be a feature of the course this year.

Those in the various federal dental corps should apply through appropriate channels. Civilians may apply to Colonel Joseph L. Bernier, DC, USA, Chief Dental and Oral Pathology Branch, Armed Forces Institute of Pathology, Washington 25, D. C.

There is no tuition for this course.

# A Clinical Study of the Effectiveness of the Roll and Charters' Methods of Brushing Teeth

*by* GLENN H. CURTIS, D.D.S., M.S.,* CLARENCE M. McCALL, JR., B.A., D.M.D., M.S.,** HOGNE I. OVERAA, L.D.S., M.S.,***

IN the broadening field of periodontal therapy the role of the toothbrush in daily oral hygiene has become increasingly important. Dental schools and practicing dentists are interested in effective methods of brushing teeth. Very little has been done to analyze the effectiveness of the existing methods, and no experimental study has been made giving evidence to support the effectiveness of any brushing method as compared with another.

It was decided, therefore, to study the relative efficiency of two methods of brushing teeth. The roll and the Charters' techniques are used in a large number of American Dental Colleges and were selected for this study.

## REVIEW OF LITERATURE

A number of pilot studies have been carried out in recent years to determine the relative efficiency of various types of toothbrushes, concerning texture, form and material of bristle.[1, 2, 3]

Other studies have dealt with the effects of brushing on the keratinization of gingival tissues. Wienmann[4] found that keratinization of the oral mucosa constituted a protective mechanism and that there were fewer inflammatory cells in the areas with greater keratinization. Orban[5] found a relationship between gingival health and keratinization of the mucosa. Stahl[6] noted an increase in gingival keratinization with increased massage and pointed out that hard natural bristles were more effective than soft ones. Robinson[7] concluded that gingival brushing increased the keratinization of the surface epithelium. Castenfelt,[8] however, was unable to find any correlation between increased keratinization and gingival health or between toothbrush massage and increased keratinization.

Gingival health as affected by type of toothbrush was studied by Hiniker and Forscher.[9] Under the conditions of this study only the six anterior teeth were used in the analysis and no attempt was made to correlate results with brushing methods.

With the intention of evaluating the cleansing effect of various dentifrices, Van Huysen and Boyd[12] used disclosing solution on the teeth and made kodachrome photographs. They found that technical considerations limited photographs to the anterior facial segments of the arches.

## MATERIALS AND METHODS

Two hundred freshmen and sophomore students from the University of Michigan School of Dentistry and Dental Hygiene were selected for this study. The students ranged in age from 18 to 31 years. The subjects were examined and the findings recorded on a special chart which had been prepared to show stained tooth surfaces and areas of gingival inflammation. The amount of inflammation in the interdental papillae and the marginal gingivae was charted on a basis of plus-1 to plus-3. Under this system a slight inflammatory involvement was graded as plus-1; a moderate inflammatory involvement as plus-2; and a severe inflammatory involvement as plus-3. All the teeth of the subjects were considered.

Following the first examination three groups were formed on a basis of the amount of gingival inflammation and the number of stained tooth surfaces. Each group represented as near as possible an equal cross section of the entire body of

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TABLE I

*Distribution of Teeth in the Experimental Groups*

| Group | Number Subjects | Total Possible Teeth Present | Total Number Teeth Missing | Total Number Teeth Present | Total Number Teeth Present |
|---|---|---|---|---|---|
| Roll | 51 | 1632 | 156 | 1476 | 28.9 |
| Charters' | 50 | 1600 | 151 | 1449 | 28.9 |
| Control | 56 | 1792 | 168 | 1624 | 29.0 |

subjects. Experimental subjects who were not able to carry out the instructions given because of illness, need for dental care, or for any other reason, were not considered in the final analysis. Case studies were completed on one hundred fifty-seven individuals.

Group I was selected to use the roll method of brushing.[13] In this technic the brush bristles are placed high in the vestibule with the bristle ends directed apically. Firm lateral pressure against the gingival tissues and teeth is maintained as the brush is moved slowly toward the occlusal plane. As the brush approaches the occlusal surface it is turned slowly so that the bristle ends touch the tooth enamel. This entire procedure is repeated six times in each area.

Group II was assigned to the Charters' method[13] in which the bristles are placed in contact with the tooth enamel and gingivae with the ends pointed at about a 45 degree angle to the plane of occlusion. Lateral and downward pressure is then placed upon the brush and the brush is vibrated gently back and forth.

Group III, the control group, was requested to continue their present method of brushing, i.e., to maintain the oral hygiene to which they were accustomed.

To assure uniformity in size and type of brush, identical brushes supplied by the same manufacturer were used throughout the study. Three brushes were given to each subject with instructions to use each brush only once in twenty-four hours and to take special care that the brushes were properly dried and ventilated after use. New brushes were distributed following the second examination.

The experimental subjects were re-examined after three weeks by the same member of the team that conducted the initial examination. No reference was made at this time to the previous record chart. Attempts were made to correct problems that any individual might be having with his assigned technique.

TABLE II

*Total Mean Amount of Inflammation*

| Group | First Examination | Second Examination | Third Examination |
|---|---|---|---|
| Roll | 40.7 | 17.9 | 8.3 |
| Charters' | 42.7 | 15.8 | 13.0 |
| Control | 33.4 | 18.6 | 15.1 |

TABLE III

*Mean Changes in Amount of Inflammation*

| Group | Upper | Lower | Total |
|---|---|---|---|
| Roll | 13.3—2.27 | 18.7—2.53 | 32.0—4.54 |
| Charters' | 14.7—2.64 | 15.4—3.17 | 30.1—5.54 |
| Control | 8.7—2.12 | 9.3—2.69 | 18.0—4.65 |

At the end of an additional thirteen weeks the final examination was conducted and findings recorded.

## RESULTS

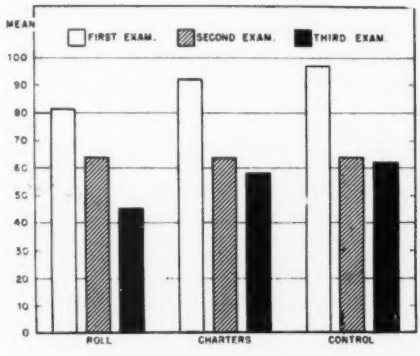
An analysis of the teeth present in each group showed the average number of teeth to be 28.9 making the total possible surfaces present (for staining) 173.4. Table I shows the distribution of teeth in the experimental groups.

Figure 1 shows the mean number of stained surfaces present in the group at each of the three examinations.

The total mean amount of inflammation in the three groups at each examination is listed in Table II.

Total mean amounts of inflammation for the marginal and papillary areas are shown in Figure 2.

The mean changes in amount of inflammation are shown in Table III.



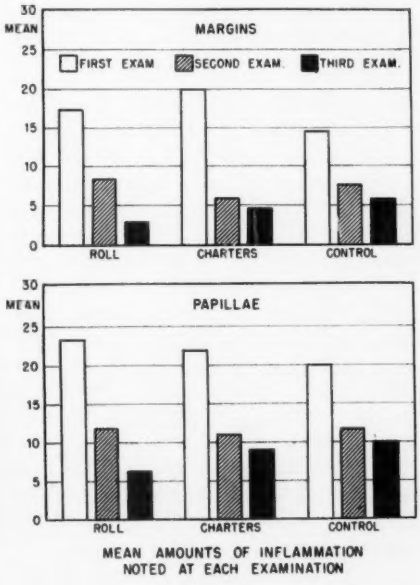

MEAN NUMBER OF STAINS NOTED AT EACH EXAMINATION

Fig. 1

## DISCUSSION

The three group exhibited a similar reduction in the mean number of stained surfaces at the second examination. The roll and Charters' groups showed a further reduction in the mean number of stained surfaces at the third examination. Although the reduction was greater than that noted in the control group the difference was not statistically significant.

The initial improvement in amount of stain in the control group may be attributed partly to the level of intelligence of the experimental subjects, and partly to a psychological factor introduced when the new brushes were distributed and the sub-




MEAN AMOUNTS OF INFLAMMATION NOTED AT EACH EXAMINATION

Fig. 2

jects informed that their mouths would be examined at given intervals. This may have been added motivation for them to maintain as high a level of oral hygiene as possible with the brushing method to which they were accustomed.

All groups showed a reduction in the mean amount of inflammation during the experimental period. The only statistically significant changes, however, occurred between the roll and control groups in the lower jaw and in the total inflammation. These changes were significant at the 5 percent level of confidence.

There was a consistently greater amount of inflammation in the lower posterior lingual and the upper posterior facial areas in all three groups at the beginning of the experiment. Those areas having the greatest mean amounts of inflammation were identical with the areas exhibiting the highest mean number of stains. This is in line with the observations of dentists everywhere, that the clean tooth is generally sound with a healthy gingiva around it, while conversely in an inflamed area one does not find 'clean teeth.' The roll method showed the most improvement in these areas at the end of the period.

The continuous reduction in the mean amount of inflammation in the roll and the Charters' groups could be due to a better understanding and application of the methods which were re-emphasized during the second examination. That inflammation in the control group was not proportionately reduced as in the other groups may be due partly to the fact that they received no instruction relative to the proper application of their method and no assistance in correcting faulty brushing habits.

## CONCLUSION

1. There was no statistically significant reduction of stain by either the roll or Charters' techniques as compared to the control group.

2. A statistically significant reduction in inflammation occurred between the roll and control groups in the lower jaw and in the total inflammation.

3. Since neither method effectively cleansed the lower anterior facial segments, dental floss or other mechanical aids are needed to supplement the toothbrush in caring for such areas.

4. To achieve optimal results from a brushing technique, the method should be carefully adapted to the needs of the individual and thorough instruction with supervised periods of drill should be given.

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# Effect on Gingiva and Occurrence of Pigmentation on Teeth Following the Topical Application of Stannous Fluoride or Stannous Chlorofluoride*

*by* JOSEPH C. MUHLER, D.D.S., PH.D., *Bloomington, Indiana*

HUMAN dental caries studies have indicated that four applications of a 2 per cent solution of stannous fluoride[1,2] or four applications of a 4 per cent solution of stannous chlorofluoride[3] are superior to four applications of either a 1 or a 2 per cent sodium fluoride solution in reducing the incidence of dental caries in children. Recently, a single application[4] of an 8 per cent solution of stannous fluoride has been shown to be significantly more effective in reducing dental caries in children than four applications of a 2 per cent sodium fluoride solution. As a result of these studies considerable interest has been shown in the chemical and physiological properties of stannous fluoride.

It has been reported that a solution of stannous fluoride at a concentration of 1000 ug fluoride per milliliter (approximately 0.4 per cent solution) will stain the teeth and gingiva if they are subsequently treated with a solution of hydrogen sulfide.[5] As a result of such *in vitro* studies, some clinicians have questioned the use of stannous fluoride as an anticariogenic agent since its use may result in detrimental esthetic effects. Based upon the clinical observation of several thousand children who have received topical applications of stannous fluoride under a variety of conditions, it has been our opinion that there were no adverse effects on the teeth, gingiva, or restorations with sufficient frequency to warrant concern. However, during the clinical investigations of stannous fluoride, numerous areas in teeth which had incipient caries became arrested, and along with this arrestment process many of the carious areas became pigmented. However, no carefully documented clinical observations were made during the clinical studies concerning pigmentation of the teeth or the effect on the gingiva, so it was considered of importance to design a study particularly for this purpose.

## METHODS

To determine his previous dental caries experience, each participating subject was examined at the initial appointment immediately following the dental prophylaxis by a dentist with the aid of a mouth mirror and explorer. During this initial visit a bite-wing radiographic examination was made. The dental caries examination was completed by charting the additional lesions observed as a result of the radiographic examination. Following the oral and X-ray examinations the children or adults were assigned at random to either the fluoride or the water group in each series on the basis of estimated caries expectancy by methods previously described.[6]

The technique of stannous fluoride, stannous chlorofluoride, or water application was as follows: following a thorough dental prophylaxis, a quadrant of the mouth was completely isolated with the aid of cotton rolls. The respective solution was applied by means of a cotton applicator, being sure that the teeth were kept moist with the solution throughout the entire four-minute treatment period. Special care was taken in all instances to keep the teeth completely isolated from saliva. Both fluoride solutions were prepared fresh each hour, and any remaining unused solution was discarded.

The state of oral hygiene was estimated by examining the entire mouth with the aid of good artificial light, dental explorers, and mouth mirrors, and by assigning arbi-

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trary values of 1, 2, or 3 to mouths with good, fair, or poor oral hygiene, respectively. The development of a method for estimating the type and extent of pigmentation is more difficult. A method is described in this paper, but it is hoped that, through additional study, other workers may help develop a more critical criterion for estimating the degree of pigmentation. The method used here, however, seems adequate for defining the nature of the problem as well as for estimating the relative frequency with which this type of pigmentation occurs and also on what teeth and surfaces it is most frequently found. Moreover, it is difficult for a person to determine whether the pigmentation in the occlusal or buccal pits and fissures is a result of stannous fluoride or is normally occurring pigmentation. Even after examining large numbers of children the exact type of pigmentation will in many cases not be readily distinguishable. However, the frequent occurrence of similar, or perhaps identical, pigmentation in teeth having no previous experience with stannous fluoride is an important phenomenon, especially if subsequent studies show that this pigmentation is associated with caries arrestment. The degree of pigmentation was estimated by examining all the teeth in the mouth after thoroughly drying each surface with air. Each value for the mouth was computed by estimating the presence of pigmentation on the various surfaces of the teeth. If it occurred on one surface a value of 1 was assigned, two surfaces a value of 2, and so forth. For purely arbitrary reasons, no mouth was counted as having pigmentation present unless 4 or more teeth were affected. In almost all instances, a value of 1 refers to the presence of pigmentation on the occlusal surface, since in over 90 per cent of the cases the pigmentation which was found on the teeth occurred on this surface. When a value of 2 was assigned, the two most frequently affected surfaces were the occlusal and the buccal (or labial).

Since the degree of pigmentation may depend on many factors, all of the subjects in this program were provided with the same dentifrice and instructed not to use any other during the experimental period in order to standardize this variable as much as possible. The dentifrice used was a standard, non-fluoride, commercial product repackaged in plain tubes. Some workers have stated that their topical fluoride studies had to be discontinued with the commercial introduction of fluoride dentifrices into the area, but by simply providing all the participants with the same control dentifrice, this objection is completely eliminated. Furthermore, such a procedure provides a much better control between groups, since all of the subjects are using the same control dentifrice.

## EXPERIMENTAL

This study was divided into 3 series. Series I was composed of 74 Indiana University freshman students, 17-34 years of age, divided into two groups. One received a single application of a 10 per cent aqueous solution of stannous fluoride, and the other, a control group, received a single application of distilled water. The fluoride or the water application was made immediately following a thorough dental prophylaxis by a dental hygienist. Six months following the initial treatment another single application of either 10 per cent stannous fluoride or distilled water was made. The duration of this experiment was twelve months.

Series II was composed of 92 children, 6 to 15 years of age, divided into two groups. Group one received a single application of an 8 per cent aqueous solution of stannous fluoride and group two a single application of distilled water. This study was continued for three months.

Series III was composed of about 200 children, 6 to 15 years of age, divided into two groups. Group one received 4 applications of a 4 per cent aqueous solution of stannous chlorofluoride and group two received 4 applications of distilled water. The first of the 4 applications of either the fluoride or water was made immediately

TABLE 1

*A Comparison Between the Oral Hygiene, the Pigmentation Index and the Dental Caries Increment in Adults or Children Receiving Topical Applications of Stannous Fluoride or Stannous Chlorofluoride.*

| Series | Treatment Group | F Conc. | No. of Subjects | Duration of Study Program | Number of Applications | Mean Oral Hygiene | Mean Pigmentation Index | Caries Increment | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Teeth | Surfaces |
| | | % | | (months) | | | | | |
| I | $SnF_2$ | 10 | 37 | 12 | 2* | 1.54 | 0.55 | 0.62 | 2.16 |
| | $H_2O$ | — | 37 | | 2* | 1.75 | 0.60 | 1.03 | 2.56 |
| | | | | | | % reduction | | 39 | 15 |
| II | $SnF_2$ | 8 | 45 | 3 | 1 | 2.17 | 0.51 | 0.33 | 0.69 |
| | $H_2O$ | — | 47 | | 1 | 2.14 | 0.29 | 0.60 | 1.32 |
| | | | | | | % reduction | | 45 | 48 |
| III | SnClF | 4 | 101 | 12 | 4 | 2.22 | 0.80 | 0.97 | 2.35 |
| | $H_2O$ | — | 107 | | 4 | 2.00 | 0.68 | 1.70 | 3.70 |
| | | | | | | % reduction | | 43 | 37 |

*Spaced 6 months apart.

following the prophylaxis and the other three were completed within a ten-day period. The first application was the only one preceded by a prophylaxis. The experimental period lasted for 12 months.

### RESULTS AND DISCUSSION

Before it is possible to determine the extent to which pigmentation of the teeth is influenced by the use of stannous fluoride, it is necessary to determine the frequency with which it occurs normally in the absence of previous contact with stannous fluoride. Some typical examples of the pigmentation are shown in the accompanying photographs. Illustrations 1 through 4 represent subjects who received topical applications of distilled water. Illustrations 1 and 2 demonstrate both the type and position of occurrence of this pigmentation. It is most frequently found in the fissures of the molars and bicuspids, and is of a brownish, sometimes almost black, color. This pigmentation is not found on smooth, non-decalcified caries-free surfaces. This is by far the most important distinction regarding this type of pigmentation, and this fact should be carefully considered when viewing similar pigmentation following the topical application of stannous fluoride. Illustration 3 demonstrates the next most frequent areas which are affected, i. e., the labial surfaces of the anteriors and the buccal surfaces of the molars and bicuspids. It is apparent again that the areas which are pigmented are those which contain

enamel faults. Illustration 4 demonstrates the next most frequent area of occurrence. It is not possible to ascertain from the illustration, but beneath the pigmentation the enamel is soft and decalcified, and operative intervention will probably be required to preserve the tooth.

Similarly, following the topical application of stannous fluoride, a pigmentation of the carious lesions which were present in the mouth at the time of the application has been noted. This is demonstrated by illustrations 5 through 7. Illustration 5 is a typical case in which the pigmentation is found on the occlusal surface of the molars. The area which is pigmented is the occlusal fissure of the second molar and not the occlusal of the second bicuspid. The second bicuspid is clinically non-carious and is not pigmented; the occlusal surface of the second molars is obviously carious and is pigmented. Illustration 6 shows the area which is next most frequently affected, i. e., the buccal pits and fissures of the molar teeth. The photograph shows the incipient lesion on the buccal surface of the left maxillary molar and the area of pigmentation. The caries-free surfaces of the bicuspids are not affected. Illustration 7 is also typical and demonstrates incipient lesions on the labial surfaces of the anteriors with subsequent pigmentation. The only portion of the tooth which is affected is that enamel which is damaged by the incipient carious process. Thus, from examining many subjects treated with stannous fluoride as well as those not experiencing contact with stannous fluoride, several general impressions have been established: (1) pigmentation frequently occurs in the control group; (2) in children receiving topical applications of either stannous fluoride or stannous chlorofluoride, there is a somewhat greater incidence of this pigmentation; (3) the pigmentation is associated with the number of carious lesions present at the time of fluoride application; (4) the pigmentation may be associated with the oral hygiene of the subjects, and (5) the non-carious surface is not affected.

Table 1 compares the oral hygiene, the pigmentation index, and the caries increment in the three series. There appears to be a numerical tendency for the group which has the poorer oral hygiene to have the higher pigmentation index as well. In the adult study, or Series I, it is seen that after 2 applications of a 10 per cent solution of stannous fluoride there was no measurable difference between the presence of this characteristic brownish pigmentation in the group receiving the stannous fluoride and in the group receiving two applications of distilled water. Perhaps one reason why no difference in pigmentation was found between the two adult groups was that most of the pits and fissures were already carious and were restored or the teeth were extracted; since this pigmentation is found predominantly in those areas little opportunity existed for its occurrence. In no instance were there any esthetic problems which could be attributed to the stannous fluoride treatment. Many of the subjects had anterior silicate or plastic restorations, and some of these were discolored, but no greater number were discolored in the group treated with stannous fluoride than in the control.

In the three-month children's study program in which a single application of 8 per cent stannous fluoride was made (Series II), there was a measurable difference between the amount of pigmentation found in the stannous fluoride and the control groups. However, since the mean pigmentation index is below 1, the pigmentation in most cases affected only a single surface; the surface most frequently affected was the occlusal surface of the molars and bicuspids (see illustration 5).

The data obtained in Series III corrobo-

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Illustrations 1, 2, 3, and 4 represent typical pigmentation which occurs in subjects who have not had previous contact with stannous fluoride. Note its color and position on the tooth.

Illustrations 5, 6, and 7 represent the color and position of typical stain which occurs following the topical application of stannous fluoride.

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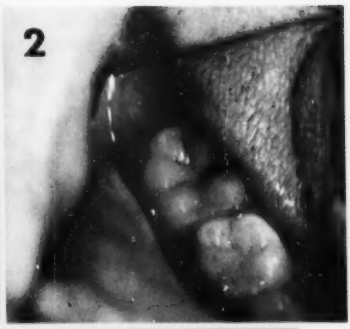
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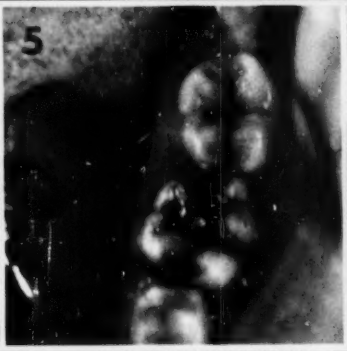
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rate the data in Series II, and suggest that those children who received 4 applications of a 4 per cent stannous chlorofluoride solution have a very slightly greater amount of pigmentation than the control group. Again, the increased occurrence of this pigmentation is confined primarily to the pits and fissures of the occlusal and buccal surfaces of the molar and bicuspid teeth.

It is our belief that this pigmentation phenomenon is associated with caries arrestment; that it, therefore, indicates the occurrence of a beneficial effect; and that it is in no way detrimental to either the health or the esthetics of the tooth or mouth as a whole. However, it is not possible to determine from these data whether the pigmented areas are associated with dental caries arrestment, since a longer period of clinical observation is needed in order to determine whether these areas are truly less susceptible to dental caries than non-pigmented carious lesions. These data do clearly indicate that the use of even a highly concentrated solution of stannous fluoride (8 to 10 per cent) is not associated with any oral esthetic problem, and the pigmentation which does occur is found predominantly in those areas of the enamel which are carious or which have pre-carious lesions. In no instance did either of the fluorides increase the prevalence of stain normally occurring in silicate or plastic restorations.

In about 5 per cent of the adults and 1 per cent of the children who received the 10 or 8 per cent solutions respectively of stannous fluoride, there was noticed a mild blanching of the gingiva. Only the free marginal areas were affected, and the effect was confined usually to the anterior region of the mouth. In a period of 8 or 10 hours the gingiva had returned to its normal appearance. The subjects experienced no pain or discomfort. It is believed that this is a result of local necrosis resulting from the precipitation of tissue protein by the high concentration of tin used, since it was always possible to demonstrate a gingivitis, of some degree of severity, in those cases in which blanching of the gingiva subsequently occurred.

The topical application of the stannous fluoride solutions resulted in a significant reduction in dental caries in each of the three series. It is interesting to note that the single application of the 10 per cent stannous fluoride solution resulted in a significant caries reduction in adults. Thus, it appears that the only effect of the fluoride treatments is one of caries prevention and that no detrimental effect is evident from such procedures.

## SUMMARY

A study was conducted in which highly concentrated solutions of stannous fluoride or stannous chlorofluoride were applied topically to the teeth of either children or adults, and compared to similar groups receiving topically applied distilled water in order to study the effect of the fluoride treatment on the pigmentation of the teeth and its effect on the oral mucous membrane. The results of these studies suggest that pigmentation of the carious areas in a tooth is a normal occurring phenomenon. Following the topical application of highly concentrated stannous fluoride solutions, there is a tendency for more pigmentation to be found in the occlusal and buccal surfaces of the molars, the occlusal surface of the bicuspid, and the labial surface of the anteriors. The non-carious tooth surface is not affected by the fluoride application to any greater degree than results following the topical application of distilled water. The peripheral portion of the interdental papilla is blanched in about 5 per cent of the cases treated with stannous fluoride if a gingivitis is present at the time of the topical application.

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# Some Epidemiological Characteristics of Periodontal Disease in a Series of Urban Populations

*by* A. L. RUSSELL, D.D.S., M.P.H.,* *Bethesda, Maryland*

THIS report describes some epidemiological characteristics of periodontal disease as observed in a series of independent urban populations. Persons from fourteen localities in New York, Maryland, Illinois, Texas, Michigan, and Colorado have been studied. Some of the groups have been re-examined at yearly intervals, so that the data summarize 31 separate studies. Included are 28,926 white persons and 1,018 negroes ranging in age from five to more than 80 years. The great majority of the persons studied were 20 years of age or younger.

While some of the data have been pooled for purposes of illustration, it must be emphasized that these were independent populations, with nothing in common except that all were urban groups and that all examinations were carried out by the author, or by examiners calibrated directly or at second hand by him. Hence, quantitative conclusions based upon these combined data should be avoided. The purpose of this paper is rather to describe a series of patterns which tended to appear consistently throughout the series of observations.

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## METHOD

All of the examinations were carried out under comparable conditions using the method of scoring previously described.[1] Under this method, each tooth is rated on a scale based upon the classical clinical signs of destructive periodontal disease—inflammation, pocket formation, and loss of function. An individual's score is the average for the teeth present in his mouth. Most persons considered to be normal, clinically, score from zero to .1 or .2; those with a clinical diagnosis of gingivitis, from .1 to 1.0; those with severe gingivitis to incipient destructive disease, from .5 to 1.9; those with frankly-established destructive disease, from 1.5 to 5.0; and those with disease in terminal stages from about 4.0 to 8.0, the maximum score.[1] In order to permit comparability between examiners, scoring is limited to flagrant and obvious signs of disease, and the data to be presented here were obtained by visual examination, without the use of x-ray. Hence, these must be considered to be minimum findings for the groups examined.

TABLE 1

*Periodontal Status of Residents in a Series of Urban Communities (Combined Data)*

| Age Group | Mean Age, Years | Number Scored | Periodontal Index Status | | | Additional Per Cent Edentulous |
|---|---|---|---|---|---|---|
| | | | Per Cent Negative | Per Cent With Pockets | Mean Score | |
| **White** | | | | | | |
| 5-9 | 7.8 | 6682 | 89.2 | 0.09 | .02 | 0.0 |
| 10-14 | 13.0 | 15922 | 74.5 | 1.0 | .09 | 0.006 |
| 15-19 | 16.3 | 4031 | 62.7 | 3.0 | .18 | 0.0 |
| 20-29 | 25.7 | 592 | 52.7 | 8.1 | .44 | 1.2 |
| 30-39 | 35.0 | 731 | 46.5 | 20.4 | .79 | 5.9 |
| 40-49 | 44.4 | 510 | 36.3 | 33.1 | 1.34 | 13.0 |
| 50-59 | 54.6 | 285 | 30.5 | 45.6 | 1.86 | 28.4 |
| 60 and over | 66.9 | 173 | 24.9 | 53.2 | 2.35 | 49.0 |
| **Negro** | | | | | | |
| 5-9 | 7.3 | 37 | 91.9 | 0.0 | .01 | 0.0 |
| 10-19 | 14.8 | 494 | 71.3 | 3.2 | .16 | 0.0 |
| 20-29 | 25.4 | 110 | 50.0 | 13.6 | .48 | 0.0 |
| 30-39 | 34.9 | 156 | 26.3 | 35.9 | 1.42 | 0.6 |
| 40-49 | 44.3 | 108 | 25.9 | 46.3 | 2.29 | 10.7 |
| 50-59 | 55.2 | 68 | 17.6 | 64.7 | 3.09 | 22.7 |
| 60 and over | 67.9 | 45 | 11.1 | 77.7 | 3.67 | 26.2 |

In addition to the mean score for a population group, two additional findings have proved of value. These are the proportions of persons free from obvious signs of disease, and the proportions of persons with one or more frank periodontal pockets.

Findings for the combined groups are shown in Table 1.

*Age*—The prevalence and severity of disease increase with age in all of the populations studied, in essentially the same manner as indicated by the combined data of Table 1.

At each age group there is a proportion of persons free of obvious signs of disease; this is true of the great majority of children nine years of age or younger. This proportion of negative persons dwindles rapidly with age until the mid-twenties, when about half of the persons seen exhibit positive signs. After this age the proportion dwindles less rapidly until, in persons aged 50 years or older, only about one-quarter of white persons and about one-tenth of negroes are free of overt disease.

The proportions of persons with disease which has advanced to the point of pocket formation increase with age in complementary fashion. About half of all whites and about two-thirds of all negroes so far seen exhibit advanced disease at or over the age of 50 years.

Periodontal scores increase with age in straight-line fashion, despite the increasing numbers of edentulous persons (who cannot be scored) at the older ages.

TABLE 2

*Relative Periodontal Scoring of White and Negro Persons Living in the Same Communities*

| Group | Number of Populations | Number Examined | Mean Age | Mean Score |
|---|---|---|---|---|
| Children enrolled in the same schools | 8 | | | |
| White | | 6867 | 12.6 | .06 |
| Negro | | 491 | 13.9 | .11 |
| Adults living in the same communities | 2 | | | |
| White | | 1982 | 39.0 | .97 |
| Negro | | 527 | 38.9 | 1.75 |

*Race*—While the findings of Table 1 indicate a higher prevalence in negroes than in whites at all ages, these data are not directly comparable, since negroes were included in only ten of the 31 population groups. The relative status of persons of the two races in these ten groups is shown in Table 2.

Negroes showed higher scores in eight of the ten populations. The two exceptions occurred in groups which supplied too few negroes—two and 23 persons, respectively—for an adequate sample.

*Sex, white*—White males and white females were represented in approximately equal numbers, with approximately the same means and distributions of ages, in all but one of the 31 studies. Composite data for males and females in the 30 white groups are shown in Table 3.

The data suggest that the sexes may differ slightly, but distinctly, in their patterns of incidence with age. The prevalence of disease is uniformly higher in males between the ages of 10 and 49 years and scores (which reflect both prevalence and severity) are uniformly higher in all males after the age of nine years. Slightly higher percentages of females with one or more periodontal pockets were observed in persons aged 19 years or younger.

Eliminating all persons with negative scores, the relative status of white males and females with obvious disease is shown in Table 4. In these groups disease, when it occurred, was more severe in females than in males up through the age of 19 years; was of about equal severity in the decade from 20 through 29 years; and was more severe in males at all ages over 30 years.

Individual findings in the 30 study groups are generally compatible with this concept. White females showed a higher proportion of persons with negative scores in 26 of the groups. Two of the exceptions occurred in very young populations (with mean age of 10.3 and 10.5 years, respectively). In the other two cases the samples were very small. In the 25 groups with mean ages under 20 years, females showed the higher proportions of persons with pocket formation in 15 groups, males in 8, with identical proportions in two. In the five groups with mean ages older than 20 years, males showed a higher proportion in four; the fifth group was very small. Males showed the higher scores in 27 of the 30 groups; in the three remaining, females exceeded males by a difference in the third

decimal place of the group scores.

Most of the observed differences were slight and relatively few were significant at the P = .01 level. If only significant differences are considered, females showed a higher proportion of negative persons in each of ten instances; males showed higher proportions of persons with pockets in each of three instances, all in groups with a mean age of 40 years or more; and males showed significantly higher scores in each of four instances.

*Sex, negro*—The limited data for negroes suggest that the same patterns may occur in that race. Most of the negro children examined by us were seen in Grand Rapids, Michigan, during the years 1952-1955, and most of the negro adults were examined in Baltimore in 1954. Data for negro males and females in these populations are shown in Table 5. In Grand Rapids children disease was more prevalent in males but tended to be more severe, when it occurred, in females. In Baltimore adults disease was both more prevalent and more severe in males.

*An interfering factor*—Comparisons between groups of children are complicated when an upper respiratory infection is endemic at the time of examination.

Initial observations in a series of continuing studies of teen-age children were made in 1952. Scores for each of these groups were significantly higher in 1953, reverting generally to the 1952 level in 1954. Study of the 1953 examination records developed the fact that an unusually high proportion of children had been noted that year as exhibiting signs of an upper respiratory infection, and that these children tended to exhibit a marked

TABLE 3

*Relative Periodontal Status of White Males and White Females in Urban Communities*

| Age Group | Mean Age, Years | Number Scored | Periodontal Index Status | | | Additional Per Cent Edentulous |
|---|---|---|---|---|---|---|
| | | | Per Cent Negative | Per Cent With Pockets | Mean Score | |
| **Male** | | | | | | |
| 5-9 | 7.8 | 3368 | 88.8 | 0.08 | .02 | 0.0 |
| 10-14 | 13.0 | 7882 | 71.0 | 0.8 | .10 | 0.0 |
| 15-19 | 16.3 | 2196 | 60.4 | 2.7 | .19 | 0.0 |
| 20-29 | 25.8 | 265 | 50.2 | 7.9 | .46 | 0.7 |
| 30-39 | 37.5 | 367 | 42.5 | 21.8 | .86 | 3.4 |
| 40-49 | 44.3 | 242 | 35.1 | 33.9 | 1.46 | 14.2 |
| 50 and over | 59.9 | 199 | 30.2 | 51.8 | 2.22 | 37.2 |
| **Female** | | | | | | |
| 5-9 | 7.8 | 3314 | 89.6 | 0.09 | .02 | 0.0 |
| 10-14 | 13.0 | 8040 | 77.9 | 1.1 | .08 | 0.01 |
| 15-19 | 16.4 | 1835 | 65.5 | 3.4 | .18 | 0.0 |
| 20-29 | 25.5 | 301 | 57.5 | 7.6 | .38 | 1.6 |
| 30-39 | 35.0 | 330 | 54.6 | 15.5 | .57 | 6.8 |
| 40-49 | 44.6 | 246 | 40.2 | 29.3 | 1.11 | 10.9 |
| 50 and over | 58.9 | 246 | 28.1 | 43.9 | 1.85 | 37.6 |

TABLE 4

*Relative Severity of Periodontal Disease in White Males and Females with Disease*

| Age group | Male | | Female | |
|---|---|---|---|---|
| | Mean Score | Per Cent With Pockets | Mean Score | Per Cent With Pockets |
| 5-9 | .22 | 0.8 | .22 | 0.9 |
| 10-14 | .35 | 2.8 | .38 | 5.1 |
| 15-19 | .48 | 6.8 | .52 | 9.8 |
| 20-29 | .92 | 15.9 | .89 | 18.0 |
| 30-39 | 1.58 | 39.8 | 1.26 | 34.0 |
| 40-49 | 2.26 | 52.2 | 1.86 | 49.0 |
| 50 and over | 3.18 | 74.1 | 2.57 | 61.0 |

and distinctive type of gingivitis. It was postulated that this factor might have been responsible for the high group scores recorded during 1953.

An opportunity to test this hypothesis directly was had early in 1955 when an epidemic of an upper respiratory infection reached its peak in one of the study schools, during the week scheduled for examination. During this entire week absenteeism due to this illness was over 40 per cent of enrollment.* Those children who were present in school and who were examined during the scheduled week were, in general, children who had not yet become ill. Most of the children examined the following week were convalescent from the illness. The distributions by age and sex were approximately equivalent for the two examination periods. Relative scoring is shown in Table 6. The difference between the groups might have occurred by accident less than once in one thousand instances.

Since that time the phenomenon has been observed consistently. The syndrome may appear after an illness too mild to prevent attendance in school. Early in convalescence the clinical picture seems to be one of a diffuse, nonlocalized gingivitis without an obvious local irritant, affecting particularly the maxillary tissues. After a few days the inflamed area contracts to a narrow, continuous line along the maxillary gingival margin from molar to molar; at this stage the mandibular tissues may appear to be normal. After a period of about ten days the mouth tends to revert to its condition prior to the illness.

In our experience, about one-third more children may show positive signs of gingivitis than expected, and expected group periodontal scores may be doubled or trebled, if a school group is examined at the peak, or early in the declining slope, of such an outbreak. No adult groups have been examined in like circumstances.

## DISCUSSION

Much data pertinent to the epidemiology of the chronic destructive periodontal diseases has been published within recent years. If care is taken to compare like matter with like, and due attention paid to the observer's methods and criteria, there is a very considerable body of agreement in this mass of material.

Gross clinical destruction of periodontal tissues, for example, has been assessed quantitatively by a number of observers. Sandler and Stahl scored the degree of bone loss, as revealed by roentgenogram, in a series of 551 white male Veterans Administration patients free of chronic disease.[2]

*An outbreak with similar clinical characteristics in a nearby school was identified as due to Influenza type B by Dr. Joseph Bell and his group in the Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health.

Stahl and Morris determined mean percentages of teeth showing recession involving the cemento-enamel junction in 1153 white males aged 17-49 years.[3] Bossert and Marks have determined the percentages of teeth affected by degenerative changes other than gingivitis, by clinical and roentgenographical examination, in 12,800 employees of the Metropolitan Life insurance company in New York.[4] When these data are plotted by age on equivalent scales with the mean periodontal scores for white persons presented in this report, the four curves are very similar in shape and characteristics. Most of the material in the report by Marshall-Day, Stephens and Quigley on 1,187 residents of Boston deals with minute changes; but their findings for average bone resorption, a gross measure, plot in equivalent fashion.[5] The five sets of data agree that the onset of chronic destructive periodontal disease may occur very early in life, and that it progresses with age in characteristic fashion.

There is, moreover, rough quantitative agreement between studies which have used somewhat comparable criteria and methods. Very similar age-prevalence curves result from the simultaneous plotting of (a) the percentages of males with active disease reported by Bossert and Marks,[4] (b) the numbers of males diagnosed as having necrotic periodontitis or periodontitis complex by Mehta, Grainger and Williams,[6] (c) the percentages of white males with alveolar bone disease among 4,274 healthy outpatients reported by Belting, Massler and Schour[7] (after their data have been recomputed for comparability to exclude edentulous men) and (d) the proportions of white males reported in this study as exhibiting one or more periodontal pockets. The agreement between these findings is quite as good as the agreement between

TABLE 5

*Relative Periodontal Status of Negro Males and Females*

| Group | Mean Age, Years | Number Scored | Periodontal Index Status | | |
|---|---|---|---|---|---|
| | | | Per Cent Negative | Per Cent With Pockets | Mean Score |
| **All persons examined** | | | | | |
| Grand Rapids, Michigan: | | | | | |
| Male | 14.8 | 200 | 73.0 | 1.5 | .12 |
| Female | 14.7 | 189 | 78.8 | 2.1 | .11 |
| Baltimore, Maryland: | | | | | |
| Male | 40.0 | 233 | 26.6 | 44.6 | 2.07 |
| Female | 38.3 | 288 | 30.2 | 34.7 | 1.51 |
| **Persons with disease** | | | | | |
| Grand Rapids, Michigan: | | | | | |
| Male | | | 54 | 5.6 | .43 |
| Female | | | 40 | 10.0 | .54 |
| Baltimore, Maryland: | | | | | |
| Male | | | 171 | 60.8 | 2.82 |
| Female | | | 201 | 49.8 | 2.17 |

TABLE 6

*Relative Periodontal Scoring of Two Groups of Children seen in a Junior High School, Maryland, 1955*

| Group | Number Examined | Mean Score |
|---|---|---|
| Examined during the week of February 1-5; mostly not ill | 798 | .05 ± .009 |
| Examined February 8; mostly convalescent from illness | 163 | .14 ± .027 |
| Difference | .09 ± .023 | |

the individual study groups observed by us and reported in this paper as pooled data. All are consistent with the conclusion of Belting, Massler and Schour, that "it can be predicted that approximately 50 per cent of all men will have roentgenographic and clinical evidence of some form of disease . . . . . by the age of 45 years."[7]

Very little evidence has been published as to possible differences between white and negro persons, and the present data must be regarded as limited in sample size and questionable as to comparability. The 470 negroes examined by Belting, Schour and Massler[7] scored only slightly higher than whites of the same ages. Whether our observed differences are due to race as race, or are associated with differences in social and economic status, must be established by further study.

Some of the reports cited dealt exclusively with males. Marshall-Day, Stephens and Quigley reported that gingival disease was more prevalent in males than in females in ten of 13 age groups.[5] Mehta, Grainger and Williams saw significantly more pockets in males than in females after the age of 35 years.[6] Bossert and Marks concluded that prevalence was slightly lower in women than in men up to the age of 55 years, and much lower after that period.[4] The suggestion in the present data, that periodontal disease may be more severe in young girls than in young boys when it does occur cannot be confirmed by these studies; they did not include younger children. In a survey of dental practice conducted by the Bureau of Economic Research and Statistics of the American Dental Association girls up through the age of 19 years apparently required more extensive treatment or extractions for periodontal disease per person with disease than did boys of the same ages, but the numbers reported as requiring treatment were small.[8] All studies agree that the difference is slight.

The clinical observation that gingivitis may be associated with upper respiratory infections obviously requires more detailed study. It is mentioned here as a factor to be considered when comparisons are to be made between two groups of children, or in the design and analysis of a clinical trial.

## SUMMARY AND CONCLUSIONS

1. This report describes some patterns of periodontal disease which have consistently appeared in a series of 31 studies of urban residents of 14 localities in six states.

2. The onset of destructive periodontal disease may occur very early in life. Advanced disease is not uncommon in teen-age children.

3. Both the prevalence and the severity of disease increase with age. At age 60 and over, about three-quarters of the white per-

sons and about nine-tenths of negroes examined showed some overt evidence of tissue damage.

4. Negroes showed a higher prevalence and a greater severity of disease than whites at all ages.

5. Disease is slightly more prevalent in males than in females. When present at all, it may tend to be somewhat more severe in young girls than in young boys. It is more severe in males than in females after the age of 20 or 30 years.

6. A short-lived gingivitis may complicate comparisons between groups of children if they are examined at the peak or early in the declining slope of an outbreak of an upper respiratory infection.

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# Periodontal Disease in Rural Children of 25 Indiana Counties

*by* E. M. BENJAMIN,* A. L. RUSSELL,** AND ROY D. SMILEY***

PREVIOUS papers have described a group method for the scoring of periodontal disease in large-scale field studies,[1] and some broad epidemiological characteristics of the disease as observed in a series of 31 studies in 14 urban localities in six states.[2] The present study was undertaken in an attempt to discover whether there might be differences in the prevalence or severity of disease which could be related to residence in a rural rather than in an urban community.

Findings are presented here for 3,880 rural children aged 9 through 20 years who were residents of 25 counties in the southern, central, and western sections of Indiana. Examinations were carried out at a 4-H camp held in the Shakamak State Park during the summer of 1955. As in former years, the camp was furnished a dental trailer and examiner by the Dental Division of the Indiana State Board of Health as part of a state-wide dental care referral program. During this period the state examiner was Mr. Thomas Sevier, an undergraduate who had completed his junior year in the Indiana University School of Dentistry. For purposes of this study the periodontal examination previously described[1] was added to the survey procedure followed in previous years.

Examinations performed by Sevier are included in the data of this report, since he proved to be adequately calibrated on test after a period of orientation. Comparability of the two senior authors, previously established, was retested at the outset. One of them was present at each examination session.

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Excellent cooperation was received from camp authorities, county leaders, and the children themselves, so that nearly every child who attended the camp during the summer was examined. Girls outnumbered boys by a ratio of about three to two.

Findings for the whole group, together with comparable data drawn from the pooled numbers of urban children of the same ages previously examined,[2] are shown in Table 1.

Up through the age of 13 years, disease in one stage or another was significantly more prevalent in these rural children (as indicated by the relative proportions negative for signs of disease) than in the urban children previously seen. Scores were significantly higher in rural children up through the age of 12 years. However, the proportions of children with advanced disease (indicated by the proportions with one or more periodontal pockets) were neither significantly nor importantly different at any point in the age range. The observed differences, in short, were due to a higher prevalence of simple gingivitis in the rural children, particularly in those aged 12 or 13 years or less.

*County scores* — Comparisons between the counties were based on findings for children aged 10-14 years, since beyond this age span neither age distributions nor the proportions of the boys and girls were uniform throughout the array. Scoring data for children of these ages, by county, are shown in Table 2.

The mean score for the whole group of 2,896 rural children summarized in Table 2 is .12, significantly higher than the mean score of .09 for the 15,921 urban children aged 10-14 years whose findings are shown in Table 1. Further, there was more variation from county to county in the Indiana children than had previously been observed between urban groups. In our experience,

TABLE 1

*Periodontal Status of 3,880 White Indiana 4-H Campers, 1955, with Common Findings for Urban Children of the Same Ages*

| *Age at Last Birthday* | *4-H Children, Indiana* | | | | *urban children* | | | |
|---|---|---|---|---|---|---|---|---|
| | *Number Examined* | *Mean Score* | *per cent of children* | | *Number Examined* | *Mean Score* | *per cent of children* | |
| | | | *negative* | *with pockets* | | | *negative* | *with pockets* |
| 9 | 113 | .10 | 70.8 | 0.0 | (rural sample inadequate for comparison) | | | |
| 10 | 585 | .10 | 72.3 | 0.0 | 1421 | .05 | 81.6 | 0.1 |
| 11 | 666 | .12 | 71.2 | 0.5 | 1689 | .07 | 78.1 | 0.5 |
| 12 | 723 | .13 | 66.9 | 0.3 | 4233 | .08 | 76.3 | 0.7 |
| 13 | 560 | .13 | 69.8 | 0.5 | 4751 | .10 | 71.9 | 1.0 |
| 14 | 452 | .14 | 70.1 | 1.1 | 3827 | .12 | 71.4 | 1.7 |
| 15 | 379 | .16 | 65.7 | 0.8 | 1773 | .18 | 62.8 | 2.7 |
| 16 | 262 | .16 | 68.7 | 2.7 | 1204 | .15 | 67.9 | 2.7 |
| 17 | 118 | .20 | 66.1 | 1.7 | (rural sample inadequate for comparison) | | | |
| 18 | 14 | .21 | 57.1 | 7.1 | (rural sample inadequate for comparison) | | | |
| 19 | 6 | .22 | 83.3 | 16.7 | (rural sample inadequate for comparison) | | | |
| 20 | 2 | .20 | 50.0 | 0.0 | (rural sample inadequate for comparison) | | | |
| | 3880 | | | | 18898 | | | |

mean scores for urban groups of these ages have ranged from .03 to .15, with scores of .12 or over occurring, generally, when a considerable proportion of the children examined had recently been ill with an upper respiratory infection.[2] Such illnesses were rare or absent in children attending the Indiana camp. While some of the county samples were small, an analysis of variance indicated that differences between the counties were due to something other than chance.

In an effort to determine whether these differences in the prevalence of gingivitis were associated with general differences in the counties from which the children came, this range of scores was compared with a series of findings taken from 1950 Census data.[3] A family of associations was discovered, based on data also summarized in Table 2.

*Percentage examined*—In order to estimate what proportion of the eligible farm children had attended the camp, it was assumed that the number of farm children aged 10-14 years resident in any county in 1955 was equivalent to the number of farm children aged 5-9 years resident in the county in 1950. These data were available in census findings.[3] The ratio of the number examined to the total estimated farm child population is shown in the final column of Table 2.

It is obvious from inspection of the data in the table that there was a tendency for the higher scores to occur in groups from those counties which sent the higher proportions of their farm children to the camp. In the eight best-represented counties, for example, about one child in nine attended camp and the group score is .14; in the others, about one child in twenty attended camp and the group score is .10—.† From the standpoint of perio-

†These and similar computations may be made directly from the data of Table 2, using the technic of weighted averages.

TABLE 2

*White Children Aged 10-14 Years in the Shakamak 4-H Camp, 1955: County Scores, Compared with Certain County Characteristics*

| County | Number Examined | Mean Score | County Characteristics, 1950 Census | | | | Per Cent 1950 cohort Examined (5) |
|---|---|---|---|---|---|---|---|
| | | | Per Cent Rural (1) | Median Years School (2) | Median Income (3) | Dentists per 100,000 (4) | |
| A | 121 | .04 | 68.1 | 8.70 | 1839 | 43 | 6.8 |
| B | 63 | .04 | 69.7 | 9.72 | 2885 | 44 | 4.6 |
| C | 34 | .05 | 76.4 | 8.62 | 1471 | 33 | 2.0 |
| D | 70 | .05 | 50.2 | 9.56 | 2796 | 34 | 5.6 |
| E | 109 | .05 | 78.1 | 10.81 | 2975 | 61 | 6.2 |
| F | 102 | .06 | 74.7 | 9.01 | 2229 | 42 | 5.8 |
| G | 32 | .07 | 77.1 | 8.15 | 1482 | 33 | 2.8 |
| H | 66 | .07 | 79.8 | 8.56 | 1747 | 33 | 5.9 |
| I | 76 | .07 | 67.3 | 8.63 | 1923 | 20 | 5.6 |
| J | 55 | .09 | 100.0 | 8.66 | 1621 | 16 | 8.7 |
| K | 70 | .10 | 66.9 | 8.30 | 1674 | 40 | 5.8 |
| L | 211 | .10 | 36.3 | 11.75 | 1942 | 48 | 9.7 |
| M | 517 | .11 | 9.0 | 11.23 | 3680 | 82 | 11.2 |
| N | 31 | .12 | 84.7 | 8.60 | 1342 | 24 | 2.4 |
| O | 244 | .12 | 25.8 | 9.09 | 2446 | 56 | 10.8 |
| P | 113 | .15 | 63.5 | 8.76 | 2438 | 49 | 6.2 |
| Q | 50 | .15 | 100.0 | 8.75 | 1678 | 9 | 4.9 |
| R | 292 | .16 | 46.1 | 8.84 | 2323 | 46 | 14.3 |
| S | 35 | .17 | 100.0 | 8.76 | 1965 | 19 | 3.2 |
| T | 175 | .17 | 77.1 | 8.84 | 1806 | 30 | 10.4 |
| U | 87 | .17 | 100.0 | 8.93 | 2046 | 32 | 6.4 |
| V | 87 | .19 | 54.0 | 8.56 | 2222 | 44 | 4.4 |
| W | 142 | .19 | 69.9 | 8.90 | 2037 | 26 | 10.9 |
| X | 70 | .19 | 100.0 | 8.85 | 2360 | 12 | 8.8 |
| Y | 134 | .19 | 64.7 | 8.72 | 2329 | 46 | 10.4 |

(1) ratio of rural farm plus nonfarm persons to total population of the country.

(2) median number of school years completed by persons 25 years of age or older in the county in 1950, adjusted according to the ratio of rural farm to rural nonfarm persons.

(3) adjusted to the proportions of rural farm and rural nonfarm persons in the country; family income.

(4) on basis of total population in county.

(5) ratio of number of children aged 10-14 years examined in 1955 to number aged 5-9 years in county in 1950, rural farm plus rural nonfarm.

dontal disease, then, the method of selection of children for camp was not comparable from county to county, and this fact must be weighed as other possible associations are considered. Two other findings are directly related to the percentages examined. In the eight counties with the lowest representation, the average of the county median incomes is $1980; in the nine middle counties, $2055; and in the

eight highest counties, $2365. For the same groups the average of the numbers of dentists per 100,000 persons in the county is 30, 37, and 43, respectively. While neither of these factors is obviously related to county periodontal scores, their association with the percentages examined suggests that a true association with county scores might appear if the bias due to non-comparable representation could be corrected.

A surface association with scores is apparent in the first two county characteristics listed in Table 2.

*Per Cent of Rural Residents*—The proportion of persons in the county classified as rural residents (the census total of rural farm plus rural nonfarm) ranged from 9.0 to 100 per cent in the counties. There was a general tendency for scores to increase as the proportion of rural residents increased. In counties with fewer than 40 per cent of rural residents the group score was .11; in counties with 40 to 80 per cent of rural residents, the score was .12; and in counties with more than 80 per cent of rural residents, the score was .15.

*Education of adults*—Census data list the median number of years of school completed by persons 25 years of age or older separately for rural farm and for rural nonfarm persons. These medians were adjusted according to the proportion of rural farm and nonfarm persons in the respective counties to yield the values shown in the table. These must be considered to be only crude approximations of the true median. On this basis, the counties may be separated into two distinct groups: those in which the median farm adult did not finish one year of high school (18 counties, with an average median of 8.7 years) and those in which the median farm adult completed one year of high school or more (7 counties with an average median of 10.5 years). The group score for all children from the 18 low counties is .14; that for all children from the 7 high counties is .09.

In addition, these social and economic findings are strongly interrelated. The greater the percentage of rural residents, for example, the lower the median incomes, and when incomes were low there was a tendency toward a smaller number of dentists per capita.

With the data available to us, it was possible to contrast urban and rural children with one of these characteristics—the median level of education of adults in the community—held to approximately equivalent terms. Examinations on urban children aged 12-14 years had been carried out in two communities where the adult level of education was about equal to that in the nine high counties. These findings, compared with children aged 12-14 years in these Indiana counties, were as follows:

| *Group* | *Number Examined* | *Median School Years, Adults* | *Mean Score* |
|---|---|---|---|
| Seven Indiana counties with median school years of 9.0 or more | 784 | 10.5 | .09 |
| Grand Rapids, Michigan (combined data, 1952-1955) | 1459 | 10.4 | .07 |
| Aurora, Illinois (1953) | 558 | 10.5 | .09 |

## DISCUSSION

While gingivitis was distinctly more prevalent in the younger rural children observed in this study than in children in a series of urban groups previously examined, it is not clear whether the difference can be ascribed to rural residence, as such, or whether it might not be explained by coincident differences not uniquely related either to rural or to urban life. The unknown factors which determined whether or not a given child attended camp obviously worked to bias county results, and the presumptive nature of the social and economic data must be kept in mind. The fact that urban and rural childen were very similar in scoring when they were drawn from communities in which the general educational level of adults was

TABLE 2

*White Children Aged 10-14 Years in the Shakamak 4-H Camp, 1955: County Scores, Compared with Certain County Characteristics*

| County | Number Examined | Mean Score | County Characteristics, 1950 Census | | | | Per Cent 1950 cohort Examined (5) |
|---|---|---|---|---|---|---|---|
| | | | Per Cent Rural (1) | Median Years School (2) | Median Income (3) | Dentists per 100,000 (4) | |
| A | 121 | .04 | 68.1 | 8.70 | 1839 | 43 | 6.8 |
| B | 63 | .04 | 69.7 | 9.72 | 2885 | 44 | 4.6 |
| C | 34 | .05 | 76.4 | 8.62 | 1471 | 33 | 2.0 |
| D | 70 | .05 | 50.2 | 9.56 | 2796 | 34 | 5.6 |
| E | 109 | .05 | 78.1 | 10.81 | 2975 | 61 | 6.2 |
| F | 102 | .06 | 74.7 | 9.01 | 2229 | 42 | 5.8 |
| G | 32 | .07 | 77.1 | 8.15 | 1482 | 33 | 2.8 |
| H | 66 | .07 | 79.8 | 8.56 | 1747 | 33 | 5.9 |
| I | 76 | .07 | 67.3 | 8.63 | 1923 | 20 | 5.6 |
| J | 55 | .09 | 100.0 | 8.66 | 1621 | 16 | 8.7 |
| K | 70 | .10 | 66.9 | 8.30 | 1674 | 40 | 5.8 |
| L | 211 | .10 | 36.3 | 11.75 | 1942 | 48 | 9.7 |
| M | 517 | .11 | 9.0 | 11.23 | 3680 | 82 | 11.2 |
| N | 31 | .12 | 84.7 | 8.60 | 1342 | 24 | 2.4 |
| O | 244 | .12 | 25.8 | 9.09 | 2446 | 56 | 10.8 |
| P | 113 | .15 | 63.5 | 8.76 | 2438 | 49 | 6.2 |
| Q | 50 | .15 | 100.0 | 8.75 | 1678 | 9 | 4.9 |
| R | 292 | .16 | 46.1 | 8.84 | 2323 | 46 | 14.3 |
| S | 35 | .17 | 100.0 | 8.76 | 1965 | 19 | 3.2 |
| T | 175 | .17 | 77.1 | 8.84 | 1806 | 30 | 10.4 |
| U | 87 | .17 | 100.0 | 8.93 | 2046 | 32 | 6.4 |
| V | 87 | .19 | 54.0 | 8.56 | 2222 | 44 | 4.4 |
| W | 142 | .19 | 69.9 | 8.90 | 2037 | 26 | 10.9 |
| X | 70 | .19 | 100.0 | 8.85 | 2360 | 12 | 8.8 |
| Y | 134 | .19 | 64.7 | 8.72 | 2329 | 46 | 10.4 |

(1) ratio of rural farm plus nonfarm persons to total population of the country.

(2) median number of school years completed by persons 25 years of age or older in the county in 1950, adjusted according to the ratio of rural farm to rural nonfarm persons.

(3) adjusted to the proportions of rural farm and rural nonfarm persons in the country; family income.

(4) on basis of total population in county.

(5) ratio of number of children aged 10-14 years examined in 1955 to number aged 5-9 years in county in 1950, rural farm plus rural nonfarm.

dontal disease, then, the method of selection of children for camp was not comparable from county to county, and this fact must be weighed as other possible associations are considered. Two other findings are directly related to the percentages examined. In the eight counties with the lowest representation, the average of the county median incomes is $1980; in the nine middle counties, $2055; and in the

eight highest counties, $2365. For the same groups the average of the numbers of dentists per 100,000 persons in the county is 30, 37, and 43, respectively. While neither of these factors is obviously related to county periodontal scores, their association with the percentages examined suggests that a true association with county scores might appear if the bias due to non-comparable representation could be corrected.

A surface association with scores is apparent in the first two county characteristics listed in Table 2.

*Per Cent of Rural Residents*—The proportion of persons in the county classified as rural residents (the census total of rural farm plus rural nonfarm) ranged from 9.0 to 100 per cent in the counties. There was a general tendency for scores to increase as the proportion of rural residents increased. In counties with fewer than 40 per cent of rural residents the group score was .11; in counties with 40 to 80 per cent of rural residents, the score was .12; and in counties with more than 80 per cent of rural residents, the score was .15.

*Education of adults*—Census data list the median number of years of school completed by persons 25 years of age or older separately for rural farm and for rural nonfarm persons. These medians were adjusted according to the proportion of rural farm and nonfarm persons in the respective counties to yield the values shown in the table. These must be considered to be only crude approximations of the true median. On this basis, the counties may be separated into two distinct groups: those in which the median farm adult did not finish one year of high school (18 counties, with an average median of 8.7 years) and those in which the median farm adult completed one year of high school or more (7 counties with an average median of 10.5 years). The group score for all children from the 18 low counties is .14; that for all children from the 7 high counties is .09.

In addition, these social and economic findings are strongly interrelated. The greater the percentage of rural residents, for example, the lower the median incomes, and when incomes were low there was a tendency toward a smaller number of dentists per capita.

With the data available to us, it was possible to contrast urban and rural children with one of these characteristics—the median level of education of adults in the community—held to approximately equivalent terms. Examinations on urban children aged 12-14 years had been carried out in two communities where the adult level of education was about equal to that in the nine high counties. These findings, compared with children aged 12-14 years in these Indiana counties, were as follows:

| *Group* | *Number Examined* | *Median School Years, Adults* | *Mean Score* |
|---|---|---|---|
| Seven Indiana counties with median school years of 9.0 or more | 784 | 10.5 | .09 |
| Grand Rapids, Michigan (combined data, 1952-1955) | 1459 | 10.4 | .07 |
| Aurora, Illinois (1953) | 558 | 10.5 | .09 |

## DISCUSSION

While gingivitis was distinctly more prevalent in the younger rural children observed in this study than in children in a series of urban groups previously examined, it is not clear whether the difference can be ascribed to rural residence, as such, or whether it might not be explained by coincident differences not uniquely related either to rural or to urban life. The unknown factors which determined whether or not a given child attended camp obviously worked to bias county results, and the presumptive nature of the social and economic data must be kept in mind. The fact that urban and rural childen were very similar in scoring when they were drawn from communities in which the general educational level of adults was

roughly equivalent suggests that rural life, in itself, is not a factor conducive to periodontal disease.

Nevertheless, there is a clear indication here that the prevalence or severity of gingival disease in children may be influenced by the social or economic conditions obtaining in the communities in which they live. This possibility should receive further study.

SUMMARY AND CONCLUSIONS

1. Using methods previously employed in studies of urban children, 3,880 white rural children aged 9 through 20 years were examined for evidence of periodontal disease. Their group scores were significantly higher than scores returned for urban children, particularly at the younger ages.

2. It is doubtful whether the observed differences can be attributed to rural life as such.

3. Associations based on presumptive evidence are presented which suggest that the prevalence or severity of periodontal disease in children may be influenced by social or economic factors.

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# *Case Report . . . .*

## Periodontal Considerations Associated with Reversible Acquired Prognathism

*by* SIDNEY S. FRIEDMAN, JR., D.D.S.,* *Memphis, Tennessee*

PROSTHO-PERIODONTIC procedures are frequently paramount to the management of periodontal pathology and often associated tempero-mandibular joint dyscrasias. The following case illustrates such an instance.

Mrs. E. D., age 42, presented for treatment of pain about the lower anterior teeth and the tempero-mandibular joint region. There was also some sensitivity to thermal change from visible exposed cervical areas and cavities. The patient was concerned about spaces forming between the lower anterior teeth and asked if anything could be done to correct this condition and the "jut of the lower jaw."

Oral examination revealed caries on the mesial and distal surfaces of the maxillary lateral incisors, posterior caries, open approximal contacts in several posterior areas, gingival inflammation, some pocket depth and the previously noted diastemas in the lower anterior region.

There was extreme prognathism and overclosure as illustrated in Figures 1 and 2. On closing in a relaxed maxillo-mandibular relationship, the incisors met in an end to end position with the pos-

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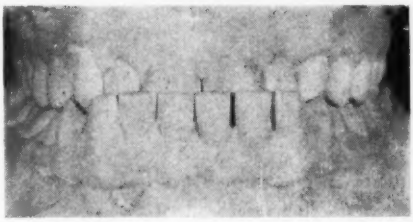

Fig. 1. Pre-operative diagnostic casts.

Fig. 2. Acquired prognathic relation. Note formation of diastemas in the lower anterior region and loss of vertical dimension, function and esthetics.

Fig. 3. Posterior function is established in physiologic centric and eccentric relationships using silver temporary treatment splints. These splints were worn several months to give assurance that a favorable prognosis could be anticipated from a permanent occlusal revision.

Fig. 4. A functional, physiologic occlusal relationship has been established in centric and the various eccentric positions.

terior teeth completely out of contact. In order to attain a functional posterior occlusion, it was necessary for the patient to go into a forced protrusive position with consequent mandibular overclosure. There was a free way space of over 5 mm. in physiologic rest position.

Radiographic examination revealed essentially a healthy alveolar bone. In several areas there was evidence of beginning widening of the periodontal membrane space and beginning loss of cortical bone.

Medical history and systemic examination findings were negative. The patient revealed that she had developed a distinct complex about her facial appearance and kept the teeth in an end to end position as much as possible. She was seriously considering maxillo-facial surgery.

The pain about the lower anterior teeth was due to oblique stresses in excess of the functional capacity of the periodontium. These stresses were brought about, because of either a tempero-mandibular joint dyscrasia, causing alteration from a normal occlusal relationship; or an acquired pathologic occlusal position, resulting in a dislocation of the tempero-mandibular joint.

The occlusal pattern was revised to restore lost vertical dimension and relieve pathologic stresses. After preparing involved teeth for full veneer coverage, a hinge axis registration was made. Utilizing these points of rotation and the infra-orbital notch as bearing points, a face bow transfer of the maxillary model was made to an anatomical articulator. Plaster occlusal registrations were taken in physiologic centric position and the mandibular model mounted using these registrations. The teeth were restored to a functional balance in physiologic centricity utilizing the above tempero-maxillary-mandibular relationships. Cusp inclines were reproduced to coincide with those generated by the patient in physiologic functional ranges.

Within a week after the detrimental stresses were relieved, the anterior diastemas closed and pain disappeared from this area and the tempero-mandibular articulation. This case offers a good prognosis as stresses are now well within the functional capacity of the periodontium. Observation after a year (Figure 4), revealed healthy supporting structures and no recurrence of the pain, inflammation or pocket formation.—*1003 Madison Ave.*

# A Consideration of Vaccine Therapy in Periodontal Disease

*by* BASIL G. BIBBY,* M.S.D., PH.D., *Rochester, New York*

DURING the first quarter of this century, there was considerable interest in the use of vaccines for the treatment of all types of dental pathology. Vaccines were prepared and used for everything from dry sockets to dental caries. However, the greatest use of vaccines was in the treatment of periodontal disease and a number of products were marketed in the United States and Europe for this purpose.

The majority of the published reports concerning the use of vaccines are favorable.[1] Whether this is the result of an all too common enthusiasm for new products or whether it reflects real clinical benefits cannot be determined at this time. However, regardless of how beneficial the vaccines were in some hands it appears that the cumulative experience of the users was not striking enough to maintain confidence in this type of therapy. Doubts which were raised[2] regarding the usefulness of vaccines in the field of medicine undoubtedly contributed to a loss of enthusiasm for the oral types. The outcome was that vaccine therapy lost ground and before long periodontal vaccines disappeared from the American market.

The possibility that vaccine therapy might have been abandoned too quickly is suggested by Appleton[1] who writes:

"A rather large body of evidence favorable to the use of bacterins (vaccines) in oral pyorrhetic infections exists. At least some of this evidence has been reported by observers, admittedly experienced and of critical spirit. Under the circumstances, the author is of the opinion that so little systematic thought and labor has as yet been given to the subject that we would be unjustified in discarding at the present time such a theoretically useful weapon as vaccine therapy."

Now that more than a quarter of a century has passed during which the possible usefulness of vaccine therapy has been ignored, it seems appropriate to re-examine the possibilities of this therapeutic approach; to consider why the procedure failed in the past and to decide whether in the light of newer bacteriological knowledge there is any justification for reconsidering its use.

Among the reasons why the vaccine treatments of thirty or more years ago were disappointing is probably that too much was expected of them. They were recommended almost entirely for use in "pyorrhea" or advanced periodontitis with active suppuration, particularly in cases which were resistant to other treatments. Apparently, it was hoped to cure these without any other treatment simply by giving a short course of intramuscular of alveolar vaccine injections. Regardless of the potency of the vaccines used, which may have been insignificant, it now seems that nothing but failure could have been expected unless local factors had been eliminated.

The nature of the vaccines used can be offered as another reason why they failed. They were usually designed to combat pyogenic organisms, such as staphylococci, which it was generally believed were actively invading and proliferating in the periodontal connective tissue and bone. This is not in keeping with our[3, 4] current concept either of the nature of the bacteria involved or the mechanism by which they attack the tissues. It should also be noted that the staphylococci at least do not produce a strong antibody response and would be unlikely to raise serum antibody levels to the point of providing protection in the periodontal tissues. However, this weakness was realized and some of the later vaccines were planned to produce a local tissue resistance by use of concentrated antigens of mixed gingival organisms.

*Eastman Dental Dispensary.

The demise of the later vaccine products was speeded by the times in which they appeared. They were placed on the market during the period in American dentistry when, in fear of "focal infection," all teeth showing any pathology were extracted forthwith without any attempt being made to encourage re-establishment of normal conditions. Since then, there has been a change in the thinking of dentists which would provide a more favorable climate for vaccine or other adjuvant therapy. Not only is there now an interest in conserving teeth and in using any necessary procedure to restore them to a healthy state, but it is also realized that if the best results are to be obtained, periodontal disease must be treated before suppuration has become established and that no therapy will give satisfactory results unless all adverse local conditions are eliminated.

Finally, it has to be suggested that all of the above reasons may be insignificant compared with the possibility that vaccine therapy of periodontal disease failed to gain full acceptance because it did not really produce any benefits and because the theory and experiment on which it was based was not scientifically sound. It is relevant, therefore, to see whether modern bacteriological concepts can be used to justify the use of vaccine treatment in periodontal disease.

In attempting a bacteriological justification of vaccine use in periodontal disease, consideration must be limited to those types of periodontal disease in which bacterial factors may be important and in which antibody response might theoretically occur in the region of involvement. This immediately places us in an area of discussion in which complete agreement is unlikely. Recent studies of the role of bacteria in periodontal disease seem to show that in gingivitis and its sequela periodontitis, tissue destruction is the result of externally placed organisms[3] elaborating products such as hyaluronidase, beta-glycuronidase and collagenase[4, 5, 6] which in turn are responsible for destruction of the periodontal tissues. It follows that if vaccines, or other agents, could strengthen the defenses of the periodontal tissues against bacteria or their products they would increase resistance and favor repair. Since, as far as we know, no attempt has been made to demonstrate antibody formation in the gingiva, there is no direct way of assessing the potential effectiveness of vaccine treatments. However, it should be noted that, theoretically at least, such effects could be produced by local treatment of the gingiva independently of the appearance of demonstrable antibodies in the serum. This conclusion is based on the knowledge tissue resistance is not necessarily associated with demonstrable antibodies and on investigations[7, 8] which demonstrated that localized areas of the skin can develop resistance to subsequent injections of pathogenic organisms. On this subject, Topley[9] concludes that the possibility that areas of skin or mucous membrane can build up a resistance to tissue destructive antigens is no longer subject to serious doubt and that "it is possible to induce an immunity which is confined to the neighborhood of the treated area and is not shared by the body as a whole." It is further suggested that this local immunity is often quite nonspecific and may be related to inflammatory involvement.

Evidence that mucous membranes can mediate in antibody reactions is found in the heightened resistance to toxin conferred by intranasal applications of toxoid[10] and the demonstration of raised antibody titres in nasal mucosa after local vaccine treatments.[11] Additional evidence appears in the quicker development of high levels of antibodies in the bronchial secretions than in the serum, after the inhalation of influenza antigens[12] and the presence of antibodies in mucous secretions of the uterus of cows in which no serum antibodies can be detected.[14] To the extent to which it might also apply to the mucous membranes of the mouth, Pierce's[13] conclusion is of interest. He states:

"Local antibodies, therefore, detected in

mucous derived from the gut, bronchi or reproductive tract may confer an immunity, particularly to localized infections of these organs which may be far more effective than that depending upon a high level of circulating antibody."

While the above conclusion has some bearing on the possibility of antibody formation by the mucous membranes of the mouth and development of localized areas of tissue immunity in the periodontium, there is little direct evidence to show that this really happens. The only pertinent findings seem to be the report[14] of development of resistance to virulent pneumococci in the buccal mucosa of rabbits following submucous injections of killed organisms and a claim[15] of increased local resistance produced in man by massaging the gums with a jelly containing bacterial antigens.

The possibility of antibody mobilization in the gingival tissues will depend to a large degree on the sites of antibody formation in the body. This is certainly not the place to enter into a discussion of this topic, but it is safe to state that recent studies[16, 17] indicate that the lymphatic system plays a dominant role and that the skin may also be involved especially if it has been primarily or secondarily stimulated with the antigen. These considerations make it seem likely that since areas of skin or mucous membrane may build up regional immunities to antigens to which they have been exposed, the gingival mucosa could react in the same way. Possibly, the lymphoid patches which have been described in the gingiva could also have some significance in this matter.

In the last analysis, the possibility that vaccine therapy could be effective in periodontal involvement must depend not upon theoretical considerations, such as the above, but on whether the right type of treatment will stimulate the mobilization in the gingiva of effective tissue defenses which might be an antibody response or some other effect in the microenvironment of the gingiva which will reduce the disease process.

The question of what type of antibody would be desirable to protect the gingiva depends upon the nature of the bacterial attack. Recent evaluations[3, 4, 5] seem to indicate that except in certain forms of acute gingivitis this is nonspecific in nature and that variations in the intensity of such nonspecific attack depends more upon changes in the numbers of bacteria or their enzymatic properties than on the appearance of new pathogenic species; that the common types of mouth cocci are the important bacterial factors and that, at least in chronic periodontal disease, any increase in the numbers of the fuso-spirochetal flora is likely to be the result rather than the cause of tissue inflammation and breakdown. All of this suggests that an antigen composed of the predominant bacterial types found in the gingival region would be most likely to stimulate the desired tissue resistance. This would be particularly true if agents such as the antigenically specific hyaluronidases[18] were part of it.

### SERIOUS QUESTION

One serious question must be raised regarding the possible benefits of a vaccine composed of mouth organisms. That is why the continued presence of the organisms in the mouth does not provide as favorable an antigenic stimulus as could be produced by the occasional administration of a vaccine made from the same organisms. If, as some clinical evidence[19] seems to suggest, treatment with such a vaccine preparation does confer benefits, there may be several explanations. One of these is that in the use of a vaccine the bacterial antigens are introduced into tissue locations which enable them to function more efficiently than they would on the surface of the mucous membranes. Presumably, this could be brought about either by injection into the gingiva or by application on a stiff toothbrush which would have a massaging or abrading effect on the epithelium producing something in the nature of an intracutaneous injection. A second possibility is that a vaccine composed of treated cells suspended in a lysate of the same bacteria

could have a much higher concentration of available antigen than would exist in any bacterial accumulation likely to occur in the mouth. A third possibility is that the treatment and lysis of bacterial cells might free from the cells antigenic or other components different from those liberated from organisms living in the oral environment.* It might be noted that some of these, such as hyaluronidase,[4] would facilitate passage of antigens into the tissues.

Obviously, these considerations are highly speculative and none of them can be considered as more than possibilities until some more definite evidence in support of them is found. However, the fact that such possibilities exist would seem to justify giving further attention to the possible value of vaccine therapy as a means of adding to the resistance to the periodontal tissues and thereby preventing or slowing down the progress of periodontal disease.

The foregoing considerations of the possible value of vaccine type therapy were prompted by a request for an evaluation of the subject by a group which reported favorable responses in certain types of gingivitis following carefully regulated use of a mixed bacterial antigen contained in a dentifrice base. Confirmatory clinical experience at the hands of personally chosen dentists gave enough significance to the original claim to justify the survey of the literature which has been presented and also the setting up of some clinical and laboratory studies to try to determine more definitely whether the "vaccine dentifrice" was effective and to see whether a rationale for antigen-antibody reactions could be established. Work along these lines is now underway in several institutions.

*It should be kept in mind that biologically active bacterial products could independently of antibody formation benefit the gingival tissue as a result of their effects on the gingival environment or the gingiva themselves.

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# Comparison of Bacterial Accumulations on Rough and Smooth Enamel Surfaces

*by* MARJORIE L. SWARTZ, B.S.* AND RALPH W. PHILLIPS, M.S.,** *Indianapolis, Indiana*

A NUMBER of investigations have studied the abrasive effects of dentifrices on tooth structure, particularly dentin.[1-5] These studies have indicated that certain dentifrices cut or abrade dentin more severely and rapidly than do others. Likewise, some of the abrasives[6] commonly employed in dentifrices and the commercial products themselves[7] exhibit a definite polishing action on enamel. Some actually abrade enamel, leaving a dull surface, while others tend to be relatively inert. Certainly a dentifrice which produces a highly polished surface is desirable from an esthetic point of view. A highly polished tooth reflects more light and appears brighter.

There is little scientific evidence, however, to indicate that a highly polished tooth surface offers any other specific advantages. For example, solubility studies have demonstrated no difference in the rates of dissolution of abraded and polished enamel.[8] It would seem logical, however, that a smooth surface might have less tendency to collect and retain debris and bacteria than a rough one. Waerhaug[9] has observed that a rough surface on dental restorations appears to facilitate the retention of bacterial plaques.

It was the purpose of this present study to investigate the accumulation and retention of debris by rough and smooth tooth surfaces in a quantitative manner. The method was an *in vitro* one which involved a comparison of the number of bacteria collected and retained by abraded and polished enamel which was subjected for a specific time to an environment containing a given number of organisms.

## PROCEDURE

In evolving a suitable test method many technical problems were encountered and a great deal of preliminary work was necessary before a workable technique was established.

There seemed to be no particular advantage in using bacteria common to the oral cavity. Serratia marcescens, an aerobic facultative organism, was selected because of the ease with which it could be cultured. Central incisor teeth were used and carefully selected to minimize cracks, crevices, and other surface irregularities. The root was removed and a stiff wire was inserted in the pulp chamber and secured by dental cement. This wire provided a means of suspending the tooth in the bacterial culture. Utilizing the entire crown for the tests did not prove feasible as it was impossible to polish or roughen all surfaces uniformly; hence, the enamel of the labial surface was cut down leaving only a raised rectangular portion (Fig. 1). This technic provided an area of controlled size which could be uniformly polished or abraded. The remainder of the tooth was then covered and sealed in such a manner that only this rectangle of enamel was exposed to the bacteria.

Both the sealing and sterilization of the tooth posed some difficulty. Cold sterilization did not prove entirely effective and it was necessary to use steam sterilization. Moist heat at a temperature of 121°C. did not produce cracks in this critical surface; therefore, the tooth was sterilized by placing it in a tube of distilled water and autoclaving for 15 minutes. The material used to cover the tooth had to be one which would withstand both heat and moisture, seal the tooth and yet be easily removed. The sealing was accomplished by a combination of two materials. Part of the wire and all surfaces except the raised enamel rectangle were first painted with a high heat aluminum paint (Fig. 1a). They were then covered by two layers of tin foil (Fig. 1b). All edges were carefully burnished with particular attention being given to those areas surrounding the enamel window. A second coat of paint was then applied to seal the foil completely (Fig. 1c).

Tests were made to check the efficiency of the seal after autoclaving by placing the covered tooth in an aqueous solution of malachite green for one hour. On removal of the covering material there was no evidence of leakage. The method was tested

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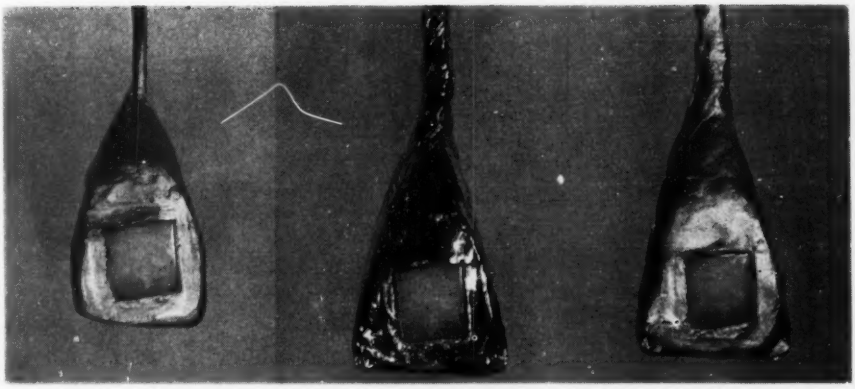

1a. 1b. 1c.

Fig. 1.

further by immersing a completely covered tooth in a bacterial broth culture for four and one-half hours. The tooth was then taken from the culture, rinsed in sterile distilled water and the foil covering removed with the aid of a sterile instrument. Following this the tooth was placed in a tube of sterile nutrient broth for four hours. After 48 hours incubation no growth was observed in this broth. Thus this technique made it possible to expose one given area of the tooth to the bacterial culture yet maintain the other surfaces sterile.

Since this study was a quantitative one involving a comparison of bacteria retained by a given area of rough or polished enamel, it was felt that the variation in the number of viable organisms in the culture might greatly influence the number of bacteria accumulated on the surfaces. Precautions were taken to minimize this variable. The broth culture was prepared by innoculating five ml. of dextrose heart infusion broth from an agar slant by means of a wire loop. This broth culture was incubated for 24 hours. At the end of this time the culture was thoroughly agitated and a 0.1 ml. aliquot was added to another 5 ml. of broth. Following incubation for an additional 24 hours, a 0.1 ml. aliquot of this culture was used to innoculate another new culture. This procedure of innoculation of new cultures was continued until termination of the study. Thus the tooth was always placed in 24 hour cultures which would contain approximately equal numbers of viable organisms. That fact was confirmed by platting the cultures and making counts. These control counts were made periodically throughout the experiment.

A highly polished enamel surface was achieved by first abrading the test area with a 4/0 abrasive paper and then by buffing on a felt wheel with Adolph Buhler's alpha polishing agent. The abraded surface was obtained by means of a #3 abrasive paper. These methods of polish and abrasion were of course exaggerated and resulted in surfaces which were smoother and rougher than those produced by commercial dentifrices. However, it was felt that the use of such extreme measures was justified. If no differences were observed it would be unlikely that the lesser differences produced by commercial products would significantly influence the accumulation of bacteria and debris under clinical conditions.

In making comparative counts of the bacterial accumulation on these surfaces, tests were made utilizing the exact same area of the same tooth—that area first being polished and then abraded. This of course eliminated individual differences in the size and number of cracks with the only variable being the type of surface exposed to the bacteria.

The actual test procedure was as follows. The test surface was prepared, sealed and the tooth sterilized. The sterile tooth was then placed in the 24 hour old culture and incubated for three hours. The foil covering was then removed and the experimental area was rinsed for 20 seconds in a stream of sterile distilled water and then immersed in a tube of sterile broth for three hours. During this period the culture media was, of course, contaminated from the bacteria which had accumulated on the enamel window. It was reasoned that the amount of contamination would bear a direct relationship to the number of organisms present on the tooth surface. At the end of the three hour period, various dilutions of this broth were made with normal saline. Duplicate nutrient agar plates were then poured.

The use of different dilutions made it possible to check the results and also to obtain plates which had an optimum number of colonies for counting. The dilutions as they correspond to the plate numbers are as follows.

| *Plate No.* | *Dilution* |
|---|---|
| 1 | None |
| 2 | 1 part in 4 |
| 3 | 1 part in 16 |
| 4 | 1 part in 64 |
| 5 | 1 part in 256 |
| 6 | 1 part in 1024 |
| 7 | 1 part in 4094 |

These plates were counted after 24 hours incubation. Additional counts were made at 48 and 72 hours but the counts remained unchanged.

All dilutions and platings were carried out according to standard bacteriological procedures. Dextrose heart infusion broth was the culture media used. The plates were poured with a 2 per cent nutrient agar which was held at a constant temperature of 42°C. Incubation of cultures and plates was done at 28° ± 3°C. The plates were counted by means of a Quebec colony counter.

## RESULTS

Six teeth were tested in the manner described and the average bacterial counts from both the rough and polished surfaces are given in Table I. Individual results revealed that in every case the plates prepared from broth in which rough surfaces had been incubated showed markedly greater counts than those prepared from broth in which polished surfaces had been immersed. This greater contamination of the broth by the abraded surface would seem to indicate a higher bacterial accumulation on that surface. Thus from these data it appears that a rough tooth surface does accumulate more bacteria than a polished one.

TABLE I

| *Plate Numbers* | *Average Bacterial Counts* | |
|---|---|---|
| | *Rough Surface* | *Polished Surface* |
| 1 | 1260 | 170 |
| 2 | 380 | 70 |
| 3 | 160 | 20 |
| 4 | 50 | 10 |
| 5 | 10 | 0 |
| 6 | 5 | 0 |
| 7 | 0 | 0 |

Although these data indicated that more bacteria accumulated on the rough tooth surface, it was not known if the bacteria would be retained any more readily if the surfaces were thoroughly brushed. Thus a series of tests were carried out where instead of merely rinsing the area upon removal from the culture, the surface was thoroughly brushed by means of a sterile motor-driven, rotary brush and sterile distilled water. The average results on a series of seven teeth appear in Table II. Again the tubes of broth in which the rough surfaces were incubated displayed higher bacterial counts than those in which the polished surfaces were immersed. It would seem from these data that not only do rough surfaces accumulate greater numbers of bacteria than polished ones but that they retain the bacteria more readily, even after vigorous brushing.

TABLE II

| *Plate Numbers* | *Average Bacterial Counts* | |
|---|---|---|
| | *Rough Surface* | *Polished Surface* |
| 1 | 1180 | 210 |
| 2 | 220 | 80 |
| 3 | 70 | 20 |
| 4 | 20 | 10 |
| 5 | 5 | 0 |

## DISCUSSION

In this series of tests it was, of course, not possible to duplicate exactly the oral conditions. It was felt however that the ease with which the bacterial organisms were collected and retained on the surfaces would be somewhat indicative of the relative facility with which stain, plaque and other debris would collect on rough and smooth surfaces in the mouth.

The differences in the number of organisms collected by the two types of surfaces was rather dramatic but the methods of polish and abrasion were likewise exaggerated. Commercial dentifrices do not produce such gross degrees of polish and abrasion as were affected here. Nevertheless it would seem logical to assume that this same phenomenon would be exhibited to a lesser degree by teeth which were brushed with a dentifrice that either abraded or polished the surface. Therefore, the use of a dentifrice which produced a smooth surface might be advantageous clinically. These experiments suggest that a polished surface would not only be desirable from an esthetic point of view but also in the maintenance of general mouth cleanliness.

## SUMMARY

An *in vitro* technique was evolved for comparing the number of bacteria collected by rough and polished enamel surfaces.

It was found that bacteria accumulated to a greater degree, per unit of time, on a rough, abraded surface than upon a highly polished surface. Bacteria were retained in greater numbers by these rough surfaces even after vigorous brushing.

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## EASTMAN DENTAL DISPENSARY

The Eastman Dental Dispensary wishes to announce the appointment of Helmut A. Zander as head of a new Department of Periodontology. Dr. Zander comes to the Dispensary from the University of Minnesota where he held the position of professor and chairman of the Division of Periodontology since 1951. Previously, Dr. Zander was professor at Tufts University in Boston. He received his D.D.S. and M.S. degrees from Northwestern University.

The Eastman Dental Dispensary will offer, under Dr. Zander's direction, a two-year course in periodontology which can, in certain circumstances, be combined with a M.S. course.

# The Distribution of *Pseudomonas Aeruginosa* in Human Saliva and Its Relationship to Gingival Health

*by* IRVING L. SHKLAIR,* PH.D., AND RICHARD W. RENN,** D.D.S., *Great Lakes, Illinois*

THE evidence that specific microorganisms are the etiologic factors in periodontal disease is not compelling, although a variety of microorganisms have been implicated.[1] It is well known that bacteria may be primary causative factors in some diseases and may act as secondary invaders in others. Numerous potential pathogens are indigenous to the oral cavity. Local and/or systemic factors may initiate periodontal disease then an otherwise harmless bacteria may secondarily invade the disturbed tissue and further contribute to periodontal destruction.

*Pseudomonas aeruginosa*, a frequent inhabitant of the oral microflorae, is a potential pathogen which can act as a primary or secondary invader. This bacteria is highly proteolytic and thrives upon necrotic tissue. Tilden[2] was able to isolate *Ps. aeruginosa* from the salivas of students with periodontal disease. She suggested that this organism may play a role in the etiology of this disease.

## MATERIALS AND METHODS

Five to 10 ml of paraffin-stimulated saliva was collected from each of the following groups of male naval personnel: (1) 57 patients, average age 19 years, with acute and subacute necrotizing ulcerative gingivitis (Vincent's infection); (2) 35 subjects, average age 27 years, with clinical evidence of periodontal pocket formation and gingival inflammation; and (3) 214 individuals, average age 19 years, with clinical evidence of normal periodontal structures.

One ml of the paraffin-stimulated saliva, collected from each individual, was placed in 25 ml of broth containing 2% peptone, 2% $MgSO_4$, 1% glycerol, 0.001% $FeSO_4$, 0.04% $KH_2PO_4$ and 1 ppm brilliant green. This innoculum was incubated at 37° C. from one to seven days. All broth solutions with evidence of bacterial growth were subcultured on agar plates which were the same composition as the broth without brilliant green.

In order to further check for the presence of *Ps. aeruginosa*, the remaining saliva was stored at room temperature for periods up to three weeks. The presence of this organism was manifested by the production of a green color in the saliva which was due to pyocyanin production by *Ps. aeruginosa*. The organism was then isolated by subculturing on the agar plates. Representative colonies were tested and the presence of *Ps. aeruginosa* was confirmed by pyocyanin production, presence of a slimy pellicle and transitory film formation, reverse swirl formation, oyster formation and growth at 42° C.[3] All but three strains were pyocyanin producers.

Periodontal pockets were cultured for the presence of *Ps. aeruginosa* with paper points utilizing the preceding methods of isolation and identification.

## RESULTS

Table I shows the distribution of *Ps. aeruginosa* among naval personnel according to gingival health. It was isolated from 7 of 57 patients (12.3%) with necrotizing ulcerative gingivitis. Five isolations were obtained from 35 patients (14.3%) suffering from inflammatory gingival changes and pocket formation. In a sample of the

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Department of the Navy, or the Naval Service at large.

TABLE I

*The Distribution of* Pseudomonas Aeruginosa *Among Naval Personnel Classified According to Gingival Health*

| *Ps. aeruginosa* | *Necrotizing Ulcerative Gingivitis* | | *Inflammatory Gingival Change and Pocket Formation* | | *Random Sample of Naval Population (Normal Paradontium)* | | *Total* | |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | No. | % |
| Ps. Positive | 7 | 12.3 | 5 | 14.3 | 33 | 15.4 | 45 | 14.7 |
| Ps. Negative | 50 | 87.7 | 30 | 85.7 | 181 | 84.6 | 261 | 85.3 |
| Total | 57 | 100.0 | 35 | 100.0 | 214 | 100.0 | 306 | 100.0 |

recruit population with normal paradontium, this organism was recovered from 33 of 214 individuals (15.4%). It is obvious that the frequency in occurrence of *Ps. aeruginosa* in each group did not differ significantly from each other. In the total of 306 cases, there were 45 salivas (14.7%) positive for this organism.

Whenever periodontal pockets were present, direct cultures were taken along with a saliva specimen. In such cases, it was found that if *Ps. aeruginosa* was present, it could be isolated from both sources simultaneously.

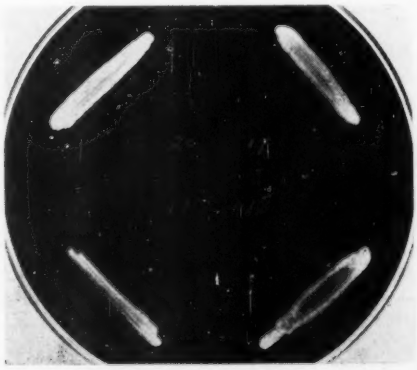

Fig. 1. Hide Powder Pour Plate Showing Typical Zones of Lysis Around **Ps. aeruginosa** isolated from a Normal Individual (No. 17) and those with Periodontal Disease (Nos. 18, 19 and 20).

Roth and Meyers[4,5] suggested that the ability of a microorganism to lyse hide powder may be a good indication of the faculty of an organism to destroy collagen in the periodontal structures. It was, therefore, decided to determine whether there was a difference in the ability of the organisms isolated to lyse hide powder. Twelve isolates of *Ps. aeruginosa* from cases with periodontal disease and 13 from individuals with normal periodontal structures were sent to Dr. Roth of the University of Kansas City, School of Dentistry to test the ability of these organisms to lyse hide powder. All strains lysed hide powder agar to approximately the same degree. There was no difference between the *Ps. aeruginosa* strains from normal individuals and those with periodontal disease. The average zones of lyses were approximately 10 mm. after 48 hours of incubation. Figure 1 demonstrates typical zones of inhibition on a hide powder pour plate around 4 separate isolates of the organism. Streak #17 is from an individual with normal periodontal structures and streaks #18, 19 and 20 are from individuals with periodontal disease.

## SUMMARY AND CONCLUSIONS

There were no significant differences in the percentage of isolates from the normal and periodontal diseased groups. *Ps. aeruginosa* was isolated from 14.7% of naval

population. The isolated organisms from both the normal and diseased groups acted similarly in their ability to lyse hide powder. It was therefore concluded that no relationship existed between *Ps. aeruginosa* and periodontal disease in the groups studied.

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# *Case Report* . . . .

## Prednisolone in the Treatment of Incapacitating Desquamative Stomatitis. Case Report

*by* EDWARD V. ZEGARELLI, D.D.S., M.S., AUSTIN H. KUTSCHER, D.D.S., AND HERBERT F. SILVERS, D.D.S., *New York, New York*

THIS report describes the effective treatment with prednisolone ointment of a patient with desquamative stomatitis[1-3] of severe and constant character which had been present for 26 years. The lesions had been relatively resistant to previously administered therapeutic agents.

Our case is also presented as evidence of a spectrum of therapeutic activity for prednisolone[4-6] differing from other corticosteroids. Cortisone acetate, administered systemically over a 2 week period in dosages of 75 mg. per day, had been only moderately effective in temporarily diminishing the objective and subjective signs and symptoms of the lesions; 2.5% cortisone acetate ointment therapy had been unsuccessful. However, as described below, prednisolone ointment 0.5% eradicated nearly all traces of the disease rapidly and repeatedly (following recurrence) after from two to seven days of therapy. Although the lesions returned after varying periods upon cessation of prednisolone therapy, the recurring lesions could be controlled at their onset by the prompt administration of prednisolone ointment.

Division of Stomatology, School of Dental and Oral Surgery of the Faculty of Medicine, Columbia University, New York, N. Y.

Prednisolone ointment (Meti-Derm) and alpha-estradiol (Progynon) were supplied by the Schering Corporation, Bloomfield, N. J.

### CASE REPORT

Initial Medical History (1931)—The patient, a well developed, well nourished, 37 year old white woman, married, with one child 9 years of age, was first seen in the Presbyterian Hospital in 1931, at which time a diagnosis was made of seborrheic eczema of the neck and temple. Mouth symptoms had been present for a few years. It was observed that the oral lesions appeared at the same time as the more severe episodes of itching of the skin with exacerbation often during menstrual periods. Skin tests for allergy were essentially negative.

Initial Dental History (1936)—The patient was referred to our clinic in 1936 complaining of watery blisters appearing on the gums at intervals. The blisters burst leaving raw, painful ulcers for several days prior to healing. The patient had received intensive routine periodontal treatment for 8 years following which the gingivae had generally improved; but the desquamative lesions continued to

recur unabated.

Initial Oral Examination (1936)—The teeth per se were non-contributory. The lesions were located generally throughout the gingivae. The marginal gingivae were swollen and cyanotic in color. There were areas which appeared dark red, raw, and hemorrhagic. Such areas were seen on the palate (left) and on the areolar gingivae from the upper left lateral incisor to the left first molar (edentulous) area and from the upper right cuspid to the right first molar region. The lower areolar gingivae evidenced large vesicles varying in location, sometimes on the lingual and at other times on the labial. These were observed, in due course, to burst, leaving shallow, desquamated, painful areas. The vesicles were transitory and varied in location. The desquamated areas in the upper jaw were constant.

Initial Biopsy. A section of gingival tissue was removed from the free gum margin in the bicuspid area of the upper left jaw. Microscopic examination revealed the epithelium to be degenerated and in some places entirely destroyed. There was a rather intense infiltration of inflammatory cells directly beneath the epithelium. The rest of the stroma showed inflammatory change also. A diagnosis was made of chronic inflammatory tissue.

Initial Diagnosis. Chronic desquamative gingivitis —vesicular variety.

Initial Treatment. Beginning in May, 1936, the patient received 10 cc of alpha-estradiol benzoate intramuscularly daily (in collaboration with the Department of Endocrinology) for 3 weeks. Each cc contained 5000 international units. The gingivae generally improved; the areas of desquamation diminished in size. During the next 10 months, approximately 45,500 international units were administered following which injections were discontinued. The mouth was relatively free of subjective and objective symptoms. A small area of desquamation in the edentulous area, upper left lateral incisor to first molar, remained.

Ten months later, the vesicular phase reappeared. The patient did not recommence treatment for an additional 5 months. For nine months thereafter (until March 1939), alpha estradiol ointment (Progynon) was applied (4,000 rat units) for thirty minutes once or twice a day and before retiring. Desquamated areas improved materially.

From then until January 1945, the patient was supplied with the ointment at frequent intervals. The oral lesions were found to be relatively controlled. During long periods from 1945-1950, the patient received intramuscular alpha-estradiol 2,000 rat units and 3 cc of crude liver once weekly (see subsequent Medical History).

Subsequent Medical History—From 1931 to 1943, the patient was seen by nearly every department of the Medical Center for one or more disturbances. In 1943 she was admitted to the hospital with evidence of a hyperchromic macrocytic anemia which responded to parenteral concentrated liver extract. The red blood cell count rose from 2,000,000 to 4,150,000, and the hemoglobin from 8.7 to 12.5 grams—accompanied by marked subjective improvement. Blood chemistry values were normal. The oral ulcerations continued.

A psychological component was described as "probably falling into a loose group of psychoses and neuroses associated with pernicious anemia and colored by the patient's pre-psychotic personality and conflicts."

The patient continued to be seen by group clinic for these important and other relatively unimportant illnesses, chiefly centering about the gastrointestinal tract, until June 1952. Full workup at that time revealed: pulse 96 and of good quality; blood pressure—128/80; head, eyes, nose were negative; there was bilateral hearing loss and tinnitus and possibly some firm enlargement of the cervical and submandibular nodes. The spine showed some evidence of thoracic scoliosis. The thorax, chest, neck, breasts, abdomen, lungs, and heart were negative. Mazzini test and Pappanicolau smear were negative; the group clinic diagnosis was chronic osteoarthritis, pernicious anemia (controlled), and undetermined gastrointestinal symptoms.

In November 1952, the patient still continued to complain of debilitating gastrointestinal pain. Her complete blood count was within normal limits. A further gastrointestinal roentgenogram series remained unchanged. The patient's pernicious anemia was relatively well controlled symptomwise throughout both the period of uncontrolled and controlled oral lesions. Also, in contradistinction to many cases of desquamative gingivitis and stomatitis in women, this patient showed signs and symptoms while still menstruating regularly.

Subsequent Oral History—As of June 1953, the oral condition remained the same as that described previously during relatively uncontrolled periods except that ulcerative lesions had spread to both cheeks, the soft palate, and centrally on the hard palate. Cortisone acetate was administered (with the collaboration of Group Clinic) orally for two weeks at a dosage of 25 mg three times daily. The mouth responded somewhat and presented an improved appearance. However, it was felt necessary to discontinue cortisone therapy owing to the possibility of systemic disturbances resulting from prolonged dosage. Several days following withdrawal of cortisone therapy, the mouth again broke out severely. It was concluded that systemic cortisone therapy could not be considered *the* definitive therapeutic agent since its administration could not be continued indefinitely without serious concern for potentially dangerous side-effects.

In November 1953, a further biopsy was taken of the ulcerated mucous membrane from the maxillary left pre-molar to the molar area on the crest of the alveolar ridge. Sections revealed: extremely vascular interlacing bundles of connective tissue focally and diffusely infiltrated by lymphocytes,

plasma cells, and neutrophils. The neutrophils were usually perivascular in distribution. The infiltration was predominantly of chronic inflammatory cells and exceedingly severe. The lining epithelium was slightly thickened, focally parakeratotic, and with elongated pointed rete pegs. The epithelium was ulcerated. The base of the ulcer was composed of chronically inflamed granulation tissue covered by a thin layer of compact fibrin.

As of the end of November 1953, and during the previous one and a half years, the patient had received the following therapeutic agents for her oral condition: Vitamin $B_{12}$ oral—20 to 50 micrograms per day for four weeks; vitamin $B_{12}$ with intrinsic factor—oral—2 per day for two courses of 3 weeks each; a dilute iodine mouthwash for 2 weeks; alpha estradiol ointment (6-10 times per day) for two weeks; calcium pantothenate 500 mg per day for 3 weeks; and cortisone acetate ointment 2.5% topically for two weeks. No significant or consistent change varying from the normal and somewhat cyclical course of her lesions was noted.

The patient's lesions remained relatively untreated and uncontrolled until January 27, 1956. At that time, prednisolone ointment 0.5% was prescribed 6 to 10 times daily (one 10 gram tube lasting for approximately one week). The response was dramatic. Within a period of two days the lesions had greatly cleared. Within one week the oral mucosa was nearly clear, marking the first such instance in many years. Topical prednisolone therapy was continued as needed during the next six months with continued complete and prompt control of those lesions which did return—these with somewhat lessened frequency. Control of subsequently recurring lesions was relatively complete within several days of their appearance, and there were comparatively long periods during which the patient was free of lesions, this in contradistinction to her previous 25 year history. Prednisolone ointment medication was continued sparingly throughout this period—applied only during periods of mild exacerbation.

As of August 9, 1956, the patient's complete blood count had remained within normal limits.

The patient was seen in our clinic periodically at monthly intervals from June through September 1957, during which time improvement was maintained. The use of prednisolone ointment was necessary for only brief periods of from 1-3 days following the onset of brief episodes of recurrence. There were no side reactions to prednisolone ointment therapy.

## DISCUSSION

On the basis of the above findings, topical prednisolone therapy seems deserving of consideration in the treatment of chronic desquamative stomatitis and gingivitis particularly should cortisone or hydrocortisone prove ineffective in the treatment of the patient. The different therapeutic spectrum of prednisolone may permit, as in this patient, beneficial therapeutic results in the presence of relative failure with topically applied hydrocortisone.

Consideration should be accorded the nearly complete absorption of prednisolone through either the oral mucous membranes in the course of topical application and subsequently (following ingestion) by the remainder of the gastrointestinal tract. Hence, topical prednisolone therapy intraorally must be considered as concurrently systemic therapy at least to the extent of the full therapeutic dosage (although minimal) employed topically.

## SUMMARY

Prednisolone ointment was found effective in the treatment of a patient with chronic desquamative stomatitis of twenty-five years' duration. Numerous therapeutic agents and regimens had previously proven relatively unsuccessful. Discontinuous alpha estradiol therapy topically and systemically had resulted in inconsistent improvement during this period. Full cortisone therapy systemically had been only partially effective in eradicating the lesions over a three week period. Cessation of cortisone therapy was followed by prompt return of the lesions with full severity. Hydrocortisone ointment topically had proven ineffective. Topical application of prednisolone, the therapeutic spectrum of which apparently differs in this instance from cortisone and hydrocortisone, has controlled the lesions at their onset, thereby allowing the patient relative freedom from disease nearly constantly.

*630 West 168th Street.*

The authors express their gratitude to Mrs. Helene W. Kutscher for her appreciated assistance.

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# *Announcements* • • • •

## ROWE SMITH MEMORIAL FOUNDATION ANNOUNCES SEMINAR DECEMBER 8-11, 1957

The fourth annual seminar of the Rowe Smith Memorial Foundation will be held December 8, 9, 10, 11, 1957 at the Fly-n-Fish Lodge on Caddo Lake, Karnack, Texas. The subject will be "Wound Healing," and the speakers, E. L. Howes, M.D., and H. B. G. Robinson, D.D.S., will bring into focus the fundamental nature of the "repair process," and the role played by vitamins, minerals, hormones, protein and antimicrobal agents. Dr. E. Cheraskin will serve as Moderator. For further information write Rowe Smith Memorial Foundation, 501 State National Bank Building, Texarkana, Arkansas Texas.

## UNIVERSITY OF MINNESOTA

The Dental and Medical Faculty of the University of Minnesota will present a course in Advanced Oral Pathology during the week of April 28 through May 2, 1958. Both lectures and pathologic slide study will be included. Microscopes will be provided. The material presented will include: neoplastic and non-neoplastic diseases of the salivary glands, neoplasms of dental origin, bone pathology, soft tissue neoplasms, dermatologic pathology, diseases of the lyphoid system, and oral roentgenographic evidence of systemic disease.

The faculty will include Dr. Robert Lukes, Armed Forces Institute of Pathology, Dr. D. C. Dahlin and Dr. Edward Stafne of Mayo Clinic, Dr. Robert Goltz of the Medical School, University of Minnesota, Dr. Anand Chaudhry and Dr. Robert J. Gorlin of the School of Dentistry, University of Minnesota.

The tuition will be $50 for the week. If room and board at the Center for Continuation Studies are desired, the cost for the week will be about $50.

Interested individuals will please contact Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis, Minnesota for further information.

# Treatment of Infectious Gingivostomatitis and Membranous Stomatitis with Terramycin Mouthwash

*by* WALTER C. STOUT, D.D.S.,* *Dallas, Texas*

INFECTIOUS gingivostomatitis and membranous stomatitis are acute infections of the oral mucosa and gingivae characterized by sudden onset, acute inflammation, bleeding, exudation, pain, and fetor oris. As these diseases progress, the mucosa becomes so extremely sore and tender that even fluids taken by mouth are very painful. Lymphadenopathy may be present, accompanied by an elevation of temperature up to 102 degrees or 103 degrees. Clinically, infectious gingivostomatitis may be diagnosed by serpiginous lesions on the attached gingivae resembling tiny circular scratches which later become small areas of necrosis covered with fibrinous exudate. In membranous stomatitis, a diphtheritic type of grayish white pseudomembrane may be found on the attached gingivae, buccal mucosa, lips, or floor of the mouth. This adherent membrane, if removed by swabbing with cotton rolls, reveals a raw, ulcerated, bleeding surface on which a new membrane will quickly reappear. Unlike oral sepsis and necrotizing ulcerative gingivitis, the oral hygiene of patients with either of these infections usually suggests good home care and regular dental attention. Extreme care should be exercised in making a differential diagnosis. Many operators initiate scaling, curettage, and prophylactic treatment at the first appointment in oral sepsis and necrotizing ulcerative gingivitis, but this treatment is contraindicated in infectious gingivostomatitis and membranous stomatitis until complete remission of the infection. Stress, debilitation, fatigue, and over indulgence are predisposing factors for infectious gingivostomatitis and membranous stomatitis. Almost every patient examined in this study gave a history including one or more of the above factors.

*Associate Professor of Periodontology, Baylor University, College of Dentistry, Dallas, Texas.

In the microscopic examination, after culturing on blood agar, large numbers of streptococcus viridans and streptococcus hemolyticus were found. Smears on slides stained with gram stain showed gram-positive cocci in pairs, chains, and groups, and a few gram-negative diplococci.

Thoma[1] describes treatment of infectious gingivostomatitis and membranous stomatitis with aureomycin or penicillin. In membranous stomatitis, he mentions the application of methylene blue, 2 per cent aqueous solution, and a mixture of crystal violet, brilliant green, alcohol, and water as treatment. Goldman[2] suggests treating infectious gingivostomatitis with one tablespoonful of white Karo syrup in a glass of hot water and in the treatment of membranous stomatitis, he uses gentian violet, and essence of Caroid with good results. Burket[3] mentions frequent hot (125°F.) sodium bicarbonate mouthwashes as beneficial and soothing and topical application of aqueous 1 per cent crystal violet or tincture of metaphen 1:200 as effective medicaments for topical use.

Fifty cases of infectious gingivostomatitis, over a period of eighteen months, were successfully treated, using 250 mg. of oxytetracycline (Terramycin) in a suspension with four tablespoonsful of warm water as a two-minute rinse after each meal and before retiring. Collings and Stout[4] have previously described this technique in the treatment of solitary and recurrent aphthae.

The only other instructions to the patient were to expectorate the solution after rinsing and to swallow any residue, to maintain a bland liquid diet for twenty-four hours and a soft diet for three days, to discard the toothbrush or brushes now in use and to resume brushing when instructed.

In forty-four of the fifty cases, patients

who had complained of even liquid diet causing pain, were much more comfortable, had less pain, and sensitivity was greatly reduced within twenty-four hours. The remaining six cases described similar results within forty-eight hours. Treatment was discontinued after seventy-two hours in every case except four. In these latter resistant cases, it was necessary to continue the rinse for four, five, six, and seven days respectively. In only two instances was it necessary to administer additional treatment. In these, where lymphadenopathy was present and the elevation of temperature did not subside in 24 hours, 600,000 units of penicillin was given intramuscularly to one patient and 250 mg. of terramycin orally every six hours for thirty-six hours to the other patient. Healing was uneventful in both cases, except for the prolonged time.

In six cases of membranous stomatitis similarly treated, five patients described a decrease in sensitivity and pain after four rinses and the remaining patient was more comfortable after two days of treatment. The course of treatment and return to clinical normal was slower and required treatment varying from four to six days. The thick, resistant, pseudomembrane possibly contributes to the delay in healing by serving as a protective covering for the organisms.

In order to test the action of terramycin mouth wash on other raw, ulcerated, and bleeding surfaces, three cases of severe chemical burn in the oral cavity were treated. The areas involved were the floor of the mouth, buccal mucosa, and lateral margin of the tongue respectively. Another case was treated after a severe reaction to a quick self-curing acrylic reliner in an upper denture which produced a necrotic and sloughing palatal mucosa within twelve hours following delivery of the denture. Diminished pain, and lessened sensitivity were noted in all four cases within twenty-four hours. As a further experiment on ulcerated lesions, six classical necrotizing ulcerative gingivitis cases were treated similarly. All six cases were less painful in one day, and there was no sign of any ulceration after three days of treatment.

## DISCUSSION AND SUMMARY

This method of treatment is rapid, nontoxic, soothing, and without systemic complications. In over sixty cases, using terramycin as a mouthwash in this treatment method, only one case of glossitis was noted. This occurred on the anterior third of the dorsum of the tongue and disappeared in three days after discontinuing treatment. The rapid analgesic effect of the terramycin is remarkable and greatly appreciated by the patients, who will often mention their greater comfort without being questioned.

Terramycin mouthwash used as a rinse is an effective agent against gram-positive diplococci, staphylococci, and streptococci viridans and hemolyticus. Patients are greatly relieved from the acute symptoms of infectious gingivostomatitis and membranous stomatitis in twenty-four to forty-eight hours. The infection is quickly brought under control and, with this treatment, infectious gingivostomatitis will be asymptomatic in three to four days and membranous stomatitis in four to six days.

Other raw, ulcerated lesions treated were cases of chemical burns and necrotizing ulcerative gingivitis. The analgesic effect was noticed in these cases within twenty-four hours and clinical healing was accelerated without the complication of secondary infection.

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# Wound Healing

*by* EDWARD LEE HOWES, M.D.,* *New York, N. Y.*

IN the process of healing, there is considerable difference between epithelization and the formation of granulations. We can no longer speak of healing in general, but must consider these two very different processes.

*Epithelization:* Regenerating epithelium springs from pre-existing epithelium. The process begins in the old epithelium by nuclear division of the cells in the basement layer. The newly formed cells wander out over the wound by amoeboid motion and then restratify to complete the architecture of newly formed epithelium. Because the predominance of nuclear division, the chemistry of epithelization is concerned more with the chemistry of nuclear division and therefore it is not surprising that anti-metabolites of nucleic acid inhibit epithelization.

*Formation of Granulations:* The formation of granulations is concerned first with the sprouting of new blood vessels and then *fibroplasia.* What initiates the sprouting of new blood vessels is not known except that vasodilation and according vasodilators do not work. The new sprouting is not inhibited by those changes that effect fibroplasia, for example, protein deficiencies except that large doses of cortisone stop both processes.

*Fibroplasia* accounts for the healing of most subsurface areas after wounding. The process originates from a "fill" of mononuclear cells or from undifferentiated connective tissue cells, not from differentiated connective tissue structure. In it, the phase of nuclear division is short, lasting only two or three days from the third to the sixth day after wounding. The rest of the process is concerned with the elaboration of fibers, first, reticulin, then collagen fibers. As the fibrils develop in fact, cells become fewer per unit of area and there are fewer blood vessels. Contractions of the surrounding uninjured tissue reduces the mass of scar tissue that must be produced to fill any defect.

The inter-relation of growth of granulations and the epithelization is very pronounced. Epithelization does not take place over exuberant granulations. New epithelial cells will extend down to depressed granulations or grow downhill over them, but the process will not change direction and grow down to and then across granulations at right angles. In some manner not understood, epithelization controls the growth of granulations, and probably there is some reciprocal relationship. For example, even when large doses of cortisone stop the growth of granulations, new granulations still are formed under the advancing edge of epithelization. Cortisone does not stop epithelization.

The *inflammatory phase* precedes the regenerative processes in healing. During the inflammatory phase, hemorrhage is arrested, bacterial invasion is resisted and dead tissue is liquefied. Ideal conditions are set up for the regeneration processes. Many enzymes are involved in the inflammatory phase. These enzymes come from the blood, from the inflammatory cells as well as from necrotizing tissues and from some of the bacteria. The inflammatory phase is prolonged by infection, by the local use of chemicals, by foreign bodies, by the presence of blood clot, by lack of adequate blood supply and for many other reasons. The duration of the inflammatory phase is much easier to prolong than to reduce. Therefore, the aim of all wound therapy must be to shorten the length of the inflammatory phase so that the regenerative processes will begin in the shortest

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Resume of Essay Given at Annual Meeting of the American Academy of Periodontology held in Atlantic City, September 27, 1956.

length of time after the wounding. Three or four days of inflammatory reaction is the best minimum that has been obtained. Once the regenerative processes are instituted in any one area, they usually progress satisfactorily. Their rates may be slowed somewhat, but they too are not stimulated by any known substances.

Wound healing in the mouth and throughout the gastro-intestinal tract takes place in a wet environment. The formation of granulations anywhere in the body takes place in a wet environment but the epithelium of the skin ideally requires a dry environment. To the contrary, the non-keratinized epithelium of the mouth is adapted to a wet environment.

There are many unsolved wound healing problems in the mouth. The blood supply of the tissues of the mouth is always excellent, yet foreign bodies from calcarious deposits are apt to form to interfere with the formation of granulations. Bacteria and salivary enzymes are always present and considerable mucus. There are even special glands to secrete mucin. Mucin contains mucoproteins, important substances in the defense of the tissues against bacterial invasion. Mucoproteins are also resistant to the alpha Amylase, the enzyme found in the saliva, and relatively resistant to trypsin. After injury mucoproteins are known to increase and to be changed in character in tissues and in the blood. Thus injured tissues would become more resistant to enzymatic action. These same types of changes should also take place in the mouth. Lysozyme that lyzes certain forms of bacteria is also present. Analyses have been made of the chemistry of the secretions of the mouth and the bacterial florae have been thoroughly investigated in relationship to healing, but what has not been taken into account is the activity of the enzymes present, and the character of the mucoproteins.

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# *Editorial Comment . . . .*

## Natural Bristles *vs.* Nylon Filaments

In an article entitled "An investigation of toothbrushes and tooth pastes used in Yugoslavia" written by Valter Krusic and abstracted in the October 1956 issue of DENTAL ABSTRACTS[1] comparisons were made of the "corrosive" effects on enamel surfaces of teeth. His conclusions were that nylon filaments cause "severe corrosion of the enamel" but no such damage was observable with natural bristles.

In studying the original article* we were disappointed to learn that the conclusions in this paper were based upon a small quantity of data obtained from a few empirical tests. In general only one of each type of specimen was employed which is certainly an inadequate number on which to form definite opinions. Apparently only one nylon brush was employed in the study and no reference was made to the bristle or filament diameter. The diameter of course will greatly influence the area of the tooth covered in the type of test which was employed.[2] Furthermore, it is obvious from the photographs taken that the lighting was not controlled and that the areas photographed were not identical in the control and experimental photograph. It seems unwise to conclude from the limited evidence presented that nylon filaments cause "severe corrosion" but natural bristles did not damage the enamel. Incidentally, a series of laboratory studies conducted by Hine et al[3] also suggest that the cleansing ability of natural brushes may be somewhat superior to nylon, but the subject must still be considered controversial.

Krusic also reports that less breakage occurred in natural bristles than in nylon filaments. The test method used by Krusic for studying the relative ability of the brushes to withstand bending and wear was an abusive test and rather unrealistic. There were no pictures of brushes before the bend tests and the pictures of the brushes after the tests are inadequate for ascertaining whether the natural bristles withstood the test better than the nylon, as is stated in the paper. These findings are not corroborated by the work of other investigators in this area. In a clinical study Wade[4] found that there was less change in the stiffness of nylon brushes than in natural ones. He noted that after prolonged use, although some of the nylon tended to bend or splay, many of the natural bristles were broken. McLane[5] found that bending of nylon appeared to be approximately ten times greater than natural bristles. In another study investigating the effect of various factors on toothbrush stiffness, after 50 minutes of mechanical brushing the natural bristles were so splayed and disheveled that a comparison stiffness of the two types of brushes could not be made.[6]

Krusic reports also that nylon wear down less than natural bristles but that nylon filaments "overtire" and break easily when used for a longer period. The wear tests employed in this study were conducted by measuring the resistance to abrasion when subjected to grinding. The author states that the nylon brush did exhibit somewhat less shortening of the bristles; however, the nylon brush had been used in a preceding test and it is possible this previous use may have influenced the results. This test would seem to be particularly heroic and has little clinical significance, as is perhaps true of many types of abrasion tests. In other toothbrushing studies involving the various dentifrices and

*Translated courtesy of Mr. Glen Millard of Lactona Inc.

common abrasives used with both nylon and natural brushes there was no detectable change in the length of either type of bristle.[7] These observations were made after periods of 300 minutes of mechanical brushing. However, wearing away of bristles is not a particularly important problem; few bristles will be worn away too much to interfere with their use. Breakage is a more important factor.

Obviously some of the results obtained in the study by Krusic are contradictory to those obtained by other workers. These differences are perhaps due to the small number of tests employed and the test methods used.

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Ralph W. Phillips
Maynard K. Hine

## THE OHIO STATE UNIVERSITY, COLLEGE OF DENTISTRY

The College of Dentistry of Ohio State University offers the following postgraduate courses for 1958:

Oral Pathology and Dental Medicine—Dr. Hamilton B. G. Robinson; March 10-14, 1958. Diseases of the hard and soft tissues will be illustrated and discussed emphasizing cause, diagnosis and treatment.

Periodontics—Dr. John R. Wilson; May 5-9, 1958. A series of lectures, discussions and clinical demonstrations on basic principles of periodontal disease emphasizing proper diagnosis, adequate treatment and essential maintenance.

Pharmacology—Dr. William E. Kampfer; May 19-23, 1958. A new course designed to refresh your knowledge of prescription writing and antibiotics and newer drugs.

For detailed information write to Postgraduate Division, College of Dentistry, Ohio State University, Columbus 10, Ohio.

# *Announcements* • • • •

## THE AMERICAN ACADEMY OF PERIODONTOLOGY TO MEET OCTOBER 31, NOVEMBER 1, 2, 1957, MIAMI, FLORIDA

*Program*

*Thursday, October 31*

A.M.

8:30- 9:00 Registration

9:00 Invocation—Edgar D. Coolidge
Necrology Report—Robert L. Dement
First Session of Forty-Third Meeting—Stanley C. Baker, President

*Reports of Standing Committees*

Committee on:

| | |
|---|---|
| Constitution and By-Laws | James P. Hollers |
| Membership | Hunter S. Allen |
| Nomenclature and Classification | Joseph L. Bernier |
| Education | Frank E. Beube |
| Research in Periodontology | Sigurd Ramfjord |
| Scientific Session | D. P. Nachazel |
| Ethics | Lowell N. Peterson |
| Local Arrangements | H. Roger Turner |
| Announcements | |

10:30 Presidential Address—Stanley C. Baker

11:00 Role of Organ Systems and Age in Periodontal Disease
Joseph Leroy Bernier, D.D.S., M.S.; FDS RCS (Eng.)
Washington, D. C.

*Luncheon*

P.M.

2:00 Stress, Body Health and the Periodontium
Perry A. Ratcliff, D.D.S., San Bernardino, California

2:45 Effect of Ovariectomy Upon the Periodontal Tissues
Irving Glickman, B.S., D.M.D., Boston, Massachusetts

3:30 Experimental Occlusal Trauma Imitating Cuspal Interferences
Balint Joseph Orban, M.D., D.D.S., Chicago, Illinois

Moderator
Sigurd Peder Ramfjord, L.D.S., M.S., Ph.D., Ann Arbor, Michigan

4:30 Open to Floor for discussion

6:00 *Friendship Hour*
di Lido Hotel—Poolside

*Friday, November 1*

A.M.

9:00 Classification of the Infrabony Pocket
D. Walter Cohen, D.D.S., Philadelphia, Pennsylvania

9:45 Reattachment and Roentgenograms
Nathan Friedman, B.S.D., D.D.S., Beverly Hills, California

10:30 Roentgenographic Interpretation
Beryl T. Ritchey, B.S., D.D.S., Colorado Springs, Colorado

Moderator
John F. Prichard, D.D.S., Fort Worth, Texas

11:30 Open to Floor for discussion

P.M.

12:15 *Round Table Luncheon*
Justin D. Towner, D.D.S. and Wallis D. Caswell, D.D.S. (Co-Chairmen)
"Metabolic Management in Stress and the Continuous Feeding Regimen"
Donald W. Smith, M.D., F.A.C.S., Professor of Clinical Surgery of the University of Miami School of Medicine, Miami, Florida

2:00 Frontiers in Bone Induction
Melvin Lionel Moss, A.B., D.D.S., Ph.D., New York, New York

2:45 Current Concepts of Bone Matrix and Factors in its Formation
C. Andrew L. Bassett, M.D., D.Sc., New York, New York

3:30 Pathology of Bone Tissue
Harry Sicher, M.D., D.Sc., Chicago, Illinois

Moderator
Saul Schluger, D.D.S., Jersey City, New Jersey

4:30 Open to Floor for discussion

7:00 *Annual Banquet*

*Saturday, November 2*

A.M.

9:00 What Is Established in Periodontology?
Sumter Smith Arnim, B.A., D.D.S., Ph.D., Houston, Texas
Robert Gottsegen, A.B., D.D.S., New York, New York
Frank M. Wentz, D.D.S., M.S., Ph.D., Chicago, Illinois
Moderator
Frank E. Beube, L.D.S., D.D.S., New York, New York

11:00 *Summation*

11:30 *Business Meeting—Election of Officers and New Members*
(Active Members Only)
Report of the President—Stanley C. Baker
Report of the Secretary—Clarke E. Chamberlain
Report of the Treasurer—James E. Aiguier
Report of Past Presidents
Dinner—Dickson G. Bell

P.M.

12:30 *Luncheon Meeting—Old and New Councils*

1:30 *Clinics*
Robert L. Reeves and Marion J. Calbeck, Co-Chairmen
"Studies on Periodontal Dressings"
Paul N. Baer, B.A., D.D.S., Bethesda, Maryland
"Sounds and Waveforms of Dental Occlusion"
Henry S. Brenman, B.S., M.S., D.D.S., Philadelphia, Pennsylvania
"The Treatment of Mobile Abutment Teeth in Oral Rehabilitation"
Maurice R. Falstein, B.S., D.D.S., Chicago, Illinois
"Histological Investigation of Gingival Regeneration in Vestibular Surgery"
Gerald P. Ivancie, M.S., D.D.S., Iowa City, Iowa
"Fibromatoses of the Gingivae"
Jesse L. Lefcourt, B.S., D.D.S., New York, New York
"Value of Radiographs in Diagnosing and Determining the Severity of Periodontal Diseases"
James S. Millsap, B.S., D.D.S., Houston, Texas
"Reactive Positioning of Teeth"
Ira Franklin Ross, D.D.S., East Orange, New Jersey
"I. The Regeneration of Periodontal Tissues Following the Reattachment Operation
II. Acceleration of Wound Healing Under the Influence of Diphenylhydantoin Sodium"
Max Shapiro, D.D.S., Beverly Hills, California

4:00 *President's Tea*
President and Mrs. Stanley C. Baker, Hosts

# INDEX

## The Journal of Periodontology, Volume 28, 1957

### JANUARY


| | | |
|---|---|---|
| Experimental Production of Periodontitis in Dogs | 7 | C. Kenneth Collings |
| Quantitative Evaluation of Therapeutic Effect of Selective Grinding | 11 | Hans R. Mühlemann<br>Hans Herzog<br>K. H. Rateitschak |
| A Measure of Tissue Response to Frequency of Toothbrushing | 17 | William R. Stanmeyer |
| Fibromata of Interest to the Periodontist | 23 | Paul N. Baer<br>Robert L. Reeves |
| Five Year Longitudinal Study of Gingival Condition of a Group of Children in England | 26 | Gilbert J. Parfitt |
| Some Observations on the Microscopic Features of Infrabony Pockets | 33 | Fermin A. Carranza, Jr.<br>Irving Glickman |
| A Review of the Epidemiology of Periodontal Diseases | 37 | Charles M. Belting |
| Orthodontic Procedures in Periodontal Therapy | 46 | Perry C. Alexander |
| Annual Reports | | |
| President's Address | 50 | Robert Kesel |
| Committee on Classification | 56 | Joseph L. Bernier |
| Resume of AAP Meeting | 58 | Harold Ray<br>A. L. Wallace |
| American Board of Periodontology | 70 | H. J. Leonard |
| Officers—1957 | 71 | |
| Letters to Editor | 78 | |


### APRIL


| | | |
|---|---|---|
| The Effects of Vertical Occlusal Trauma on the Periodontium of Protein Deprived Young Adult Rats | 87 | S. S. Stahl<br>S. C. Miller<br>E. D. Goldsmith |
| The Temporomandibular Joint and Occlusion | 97 | L. Laszlo Schwartz |
| Repositioning and Increasing the Zone of Attached Gingiva | 106 | Arnold A. Ariaudo<br>Howard A. Tyrrell |
| Gingivitis in Young Adult Males: Lack of Effectiveness of a Permissive Program of Toothbrushing | 111 | Maury Massler<br>Henry M. Rosenberg<br>William Carter<br>Isaac Schour |
| Cementum and Dentine Implants in a Dog and a Rhesus Monkey | 125 | Erwin M. Schaffer |
| Histochemistry of Calculus Formation | 132 | Irwin D. Mandel<br>Barnet M. Levy<br>Bernard H. Wasserman |
| Staining of Resin and Silicate Restorations by Topically Applied Solutions of Stannous Fluoride | 138 | Judith F. Hine<br>Marjorie L. Swartz<br>Ralph W. Phillips |
| Use of Demerol and Scopolomine for Patient Relaxation in Occlusal Equilibration | 144 | Frank T. Scott |
| Healthy Periodontium in Chronically Ill Patients | 147 | Irwin Walter Scopp |
| Poem | 154 | |
| Book Review | 137 | |
| Case Report . . . Allergic Reaction to Periodontal Pack | 151 | Irving Romanow |
| Reports | | |
| Ad Hoc Committee | 158 | |
| William J. Gies Foundation | 165 | |
| Letters to the Editor | 167, 168 | |

## JULY


| | | |
|---|---|---|
| Extension of Exudate into Supporting Structures of Teeth in Marginal Periodontitis | 175 | Henry M. Goldman |
| Bone Implants in Periodontal Diseases: A Further Study | 184 | W. G. Cross |
| Clinical and Roentgenographic Studies of Trauma from Occlusion | 192 | Ulf Posselt and Ollie Maunsbach |
| Case Histories | 196 | Myer D. Wolfsohn and Robert S. Wolfsohn |
| The Infrabony Technique as a Predictable Procedure | 202 | John Prichard |
| A New Method for the In Vitro Production of Artificial Calculus | 217 | S. W. Leung |
| Healing of Human Periodontal Tissues Following Surgical Detachment from Non-Vital Teeth | 222 | Melvin L. Morris |
| Tissue Reaction to Metal Wires in Healthy Gingival Pockets | 239 | Jens Waerhaug |
| The Value of Step-Serial Sectioning in the Morphologic Study of Human Periodontal Disease | 248 | Harold R. Stanley Jr. |


## OCTOBER


| | | |
|---|---|---|
| Experimental and Histological Investigation of Gingival Regeneration in Vestibular Surgery | 259 | Gerald P. Ivancie |
| The Application of the Ultrasonic Dental Unit to Scaling Procedures | 264 | Walter N. Johnson John R. Wilson |
| Is Prophylactic Occlusal Adjustment a Justifiable Procedure? | 272 | Robert L. Reeves |
| A Clinical Study of the Effectiveness of the Roll and Charters' Methods of Brushing Teeth | 277 | Glenn H. Curtis Clarence M. McCall, Jr. Hogne I. Overaa |
| Effect on Gingiva and Occurrence of Pigmentation on Teeth Following the Topical Application of Stannous Fluoride or Stannous Chlorofluoride | 281 | Joseph C. Muhler |
| Some Epidemiological Characteristics of Periodontal Disease in a Series of Urban Populations | 286 | A. L. Russell |
| Periodontal Disease in Rural Children of 25 Indiana Counties | 294 | E. M. Benjamin A. L. Russell Roy D. Smiley |
| Periodontal Considerations Associated with Reversible Acquired Prognathism | 298 | Sidney S. Friedman |
| A Consideration of Vaccine Therapy in Periodontal Disease | 300 | Basil G. Bibby |
| Comparison of Bacterial Accumulations on Rough and Smooth Enamel Surfaces | 304 | Marjorie L. Swartz Ralph W. Phillips |
| The Distribution of Pseudomonas Aeruginosa in Human Saliva and Its Relationship to Gingival Health | 308 | Irving L. Sinklair Richard W. Renn |
| Prednisolone in the Treatment of Incapacitating Desquamative Stomatitis | 310 | Edward V. Zegarelli Austin H. Kutscher Herbert F. Silvers |
| Treatment of Infectious Gingivostomatitis and Membranous Stomatitis with Terramycin Mouthwash | 314 | Walter C. Stout |
| Wound Healing | 316 | Edward Lee Howes |
| Editorial | 318 | |
| Announcements | 263, 271, 276, 293, 307, 313, 319- | |